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Table of Contents.

The Beattie-Smith Lectures.[1]

(UNIVERSITY OF MELBOURNE.)

INSULIN AND "CARDIAZOL" IN THE TREATMENT OF THE PSYCHOSES.

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LECTURE I: INSULIN TREATMENT.

I SHOULD like, first of all, to express my appreciation of the honour of being invited to deliver the Beattie-Smith lectures for 1940. So many distinguished psychiatrists have preceded me in this lectureship, that it was with some misgivings that I heard of my appointment, and it was only the knowledge that I had been fortunate enough to have had some experience in these new methods of treatment that induced me to appear before you tonight.

The late Dr. Beattie-Smith retired from mental hospital practice before I was born, and although in the intervening years psychiatry has made many advances, we have not yet achieved the object that Beattie-Smith had in mind when he endowed these lectures—that is, the better education of the public and the profession in psychiatric matters. The attitude of the general public to mental illness is still an unenlightened one, and I regret to say that in some respects the attitude of the medical profession is little better. In a leading article in a recent issue of *The British Medical Journal*, it was suggested that as a war time economy measure the insulin treatment of mental illness be prohibited, and this article actually suggested government intervention to prevent what it termed "a serious wastage of valuable insulin". Now it may be necessary to economize in the use of certain drugs to further the war effort; but I think it is typical of our attitude to psychiatry that the first step in this direction should be an attempt to deprive psychotics of a really valuable therapeutic measure, instead of beginning on some of the relatively useless drugs we are all so fond of prescribing. When the medical profession itself adopts this attitude, we can hardly wonder at the attitude of the general public, and I think there is a real need today for more Beattie-Smiths to champion the cause of psychiatry.

As you know, the subjects to be discussed in these lectures are insulin and "Cardiazol" treatments of mental illness. They are both large subjects, and I cannot hope to cover them completely; however, I shall try to deal with the more important and more interesting points. Tonight I shall discuss insulin treatment, while "Cardiazol" treatment and the results of both forms of treatment will be considered in the next lecture.

Introduction of the Treatment by Sakel.

The insulin treatment of schizophrenia was introduced by Dr. Manfred Sakel, of Vienna. In 1928 Sakel used small doses of insulin in an attempt to lessen the deprivation symptoms of morphine addicts, and later he tried to use it in other cases of excitement. Quite by accident some patients showed a deeper hypoglycæmic reaction than was intended, and Sakel noted that these accidents seemed to produce a definite improvement in the mental state. The treatment was extended to patients suffering from

[1] Delivered at Melbourne on November 18 and 25, 1940. The lectures were illustrated with cinematograph films.

schizophrenia, and gradually deeper and deeper hypoglycæmic states were produced.

In 1934 Sakel published a description of his method, in which he advocated the regular production of a deep hypoglycæmic coma. A small initial dose of insulin was given, and this was increased day by day until the dose sufficient to produce coma was reached. The coma was terminated by the administration of glucose, and the treatment was given every day except Sundays until coma had been produced as many as 60 or 80 times.

Insulin had been used in the psychoses for some time before the advent of Sakel's method; but Sakel's method differs in that it deliberately produced deep hypoglycæmic states, which previous workers had regarded as dangerous and undesirable.

It has been said that once in the lifetime of even the most humble medical practitioner, certain facts will be presented to him in such a fashion as to give him an inkling of the truth of what may be a baffling and unexplained disease. That opportunity was offered to Sakel, and he seized it greedily; and I think we should pay a tribute to his courage and skill in introducing a treatment which to its originator must have presented many difficult and alarming problems.

Technique.

The technique of insulin treatment has changed little since its original introduction by Sakel. The patient is examined both physically and psychiatrically to determine his suitability for the treatment.

On the first day no breakfast is allowed, and at 7 a.m. 20 units of insulin are injected intramuscularly. The patient remains in bed throughout the morning and is permitted to read, knit or smoke within reason. This small dose probably has no effect, and at midday a small glucose drink is given; soon afterwards the patient has a meal with an adequate carbohydrate content. In the afternoon he leads the normal hospital life, according to his mental condition, and at 6.30 p.m. he has his evening meal and soon after goes to bed.

On the second day the dose of insulin is increased to say 40 units; again there is little effect, and at midday a glucose drink is given.

On successive days the dose is further increased, until on about the fifth day after a dose of about 100 units signs of mild hypoglycæmia appear, and at midday the patient has to be supported to drink his glucose.

On following days the dose is again increased, signs of deeper hypoglycæmia appear, and at midday the patient is too drowsy to drink the glucose, which consequently is given by means of a nasal tube. Although unable to drink, the patient is not comatose and usually resists the passing of the tube.

For the next day or two the dose is increased further, until on about the ninth day after a dose of 150 to 200 units deep coma develops, usually about three hours after the injection of insulin—that is, at the end of the third hour of hypoglycæmia. It is customary to describe the period from the injection of insulin to the termination of the coma as the period of hypoglycæmia; and typically the patient becomes drowsy during the second hour of hypoglycæmia, the coma supervenes during the third or fourth hour, and glucose is given to terminate the coma during the fifth hour of hypoglycæmia.

The dose of insulin which is necessary to produce a coma is known as the coma dose, and, as we have seen, it is arrived at by starting with a small safe dose which is gradually increased day by day until coma supervenes. This coma dose is repeated every day except Sunday until the maximum improvement in the patient's mental state is obtained, or until the treatment is abandoned as hopeless. The smallest dose which produced coma at the Mental Hospital, Mont Park, was 10 units, the patient being a rather wasted, arteriosclerotic woman aged 55 years; the greatest dose required to produce coma was 600 units.

Commonly the patient develops some degree of sensitivity to insulin, so that the original coma dose may be gradually reduced without effect on the depth of the coma produced. Sometimes this degree of sensitivity is surprising; for example, I have seen an original coma dose of 400 units gradually reduced to 150 units, the latter dose producing quite a deep coma. On the other hand, the patient may become tolerant to insulin, so that the coma dose has to be constantly increased in order to produce any considerable depth of coma, and, as will be noted later, this procedure is not without risk. Furthermore, the response to a given dose of insulin may vary considerably from day to day: it is not uncommon to see a patient have a satisfactory coma regularly after a given dose of insulin and then suddenly after the same dose to lie awake all the morning and be able to take his glucose drink at midday. An obvious explanation is that the patient has had access to some food or sweets just before the injection; but in many of these cases it has been possible to exclude any such possibility.

It has been frequently noted that the coma on Monday is frequently lighter than usual; this is presumably due to the fact that on the preceding day, Sunday, no treatment is given, and the carbohydrate reserves of the body are restored.

Relatively few patients pass quietly through a state of increasing drowsiness into coma; they usually show a number of interesting and sometimes alarming manifestations of the increasing hypoglycæmia. These hypoglycæmic manifestations will be mentioned later, and at present I shall discuss the actual coma.

There are no definite criteria by which one can detect the presence or the depth of hypoglycæmic coma, but the following three signs seem to me the most useful. Firstly, the patient is insensitive to external stimuli; for example, pressure over the supraorbital nerve, which is normally quite painful, evokes no response. Secondly, there is complete muscular relaxation similar to that seen under general anæsthesia. Thirdly, the corneal reflex is absent. As a general rule, unless these three signs are present, the patient is not in coma; but one does see occasionally what appears to be a dangerously deep coma in which the corneal reflex is not completely abolished.

In addition to these signs there are others which are helpful. The plantar reflex is usually extensor in type, although this is by no means constant; there may be no response in an obviously deep coma, and not infrequently Babinski's reflex is present in a patient who is only semi-comatose. The condition of the pupils is helpful, and it is similar to that found during general anæsthesia; but as in the latter condition, it may be misleading. Usually during the coma the pupils are of moderate size and react to light rather sluggishly; during deep coma they tend to become fixed. Dilated, fixed pupils are regarded as a danger sign and an indication for immediate termination of the coma by the intravenous administration of glucose; however, one does occasionally see large, fixed pupils in a patient with a good pulse, good colour and normal respirations.

The depth of the coma is determined by the severity of the various hypoglycæmic manifestations to be discussed later, and most important of all by the general appearance of the patient. This may not sound very scientific, but just as in general anæsthesia the anæsthetist may remove the mask because he vaguely feels that the patient's condition is not very satisfactory, so in hypoglycæmic coma the mere look of the patient may suggest the administration of a small quantity of glucose just to be on the safe side.

The comatose state is permitted to continue for one to one and a half hours, provided the period of hypoglycæmia does not exceed five hours. Therefore, if the insulin is given at 7 a.m. and the coma develops at 10 a.m., it may be permitted to continue until about 11.30 a.m. If the coma develops late, say at 11.30 a.m., it must be terminated soon after, otherwise the five-hour period of hypoglycæmia which ends at 12 noon will be exceeded.

Termination of the Coma.

It is now necessary to consider the various methods which may be used to terminate the coma.

The usual method is by giving glucose through the nasal tube. The tube is lubricated with glycerin, paraffin or similar lubricant and passed into one nostril and down into the stomach. When the tube has been passed it is essential to ensure that it has entered the stomach and not the lung. This is done by aspirating gastric juice and testing it with blue litmus paper; if no gastric juice can be obtained, a syringeful of air is forced down the tube whilst the epigastrium is being auscultated; air can nearly always be heard entering the stomach. If both these procedures fail, it should never be taken for granted that the tube is in the stomach; the glucose solution should be given by the intravenous route. This may seem an unnecessary precaution, but it should be remembered that it is quite easy to pass the tube into the trachea of an unconscious patient, and although when this happens one usually gets an indication that something is amiss, it is quite possible for a patient to continue to breathe quietly and maintain a good colour with the tube in the trachea. The risk of allowing glucose solution to enter the lungs must of course be avoided at any cost.

When the position of the tube in the stomach has been proved, about 350 cubic centimetres of 50% glucose solution—that is, about 175 grammes of glucose—are given through the tube. It is not necessary to vary this amount of glucose, since, irrespective of the dose of insulin, it is sufficient to wake the patient and keep him awake until lunch time. Within ten minutes to half an hour of the giving of the glucose the patient gradually awakes from the coma. This awakening is often unpleasant and may be accompanied by restlessness, shouting and confusion—a condition very similar, in fact, to that seen in recovery from a general anæsthetic. On awakening, the patient has a rub down, his night clothes and bed clothes are changed and he remains in bed until lunch, after which he may get up and follow any occupation suitable to his mental condition.

The second method of terminating the coma is by the intravenous administration of glucose. About 60 cubic centimetres of a 33% solution—that is, about 20 grammes—are given intravenously and the patient awakes almost immediately without the unpleasant symptoms which accompany the nasal tube method. Providing the coma has not become irreversible, a condition to be described later, 20 grammes of glucose given intravenously will always terminate it, irrespective of the dose of insulin which has been given. The intravenous route is used in various emergencies, when the routine nasal tube method has failed to arouse the patient, or when the awakening brought about by the nasal tube feed is associated with particularly unpleasant symptoms or much nausea. It gives a much more pleasant and smoother awakening and has much to recommend it, and the tendency at Mont Park it to use it more and more. Some patients find glucose very nauseating, and in such cases the intravenous route of administration may be used as a routine method.

The third method of terminating the coma is by the administration of glucose intramuscularly. It is used for very restless patients, when the nasal tube method or the intravenous administration would be difficult, or when the nasal tube method fails to awaken the patient, and the veins are small, so that it is desirable to preserve them against an emergency. There is a theoretical objection to the intramuscular use of glucose; it is supposed to be irritating to the tissues and to lead to sloughing. However, at Mont Park a 15% solution of glucose has been given intramuscularly on numerous occasions and I have never seen the slightest harm result.

Theoretically, adrenalin could be used to terminate the coma; in my opinion it is dangerous and has no place in an insulin clinic.

Hypoglycæmic Manifestations.

It has previously been mentioned that although some patients pass quietly through a state of increasing somnolence into the hypoglycæmic coma, a majority show, either as they approach the coma or during the coma, a variety of interesting manifestations of the hypoglycæmia. These manifestations of hypoglycæmia are due to the effects of insulin on the central nervous system and the autonomic nervous system; but many of the hypoglycæmic syndromes are obviously the result of the insulin effect on both systems, and so their classification on this basis is unsatisfactory.

These hypoglycæmic signs do not appear in any regular sequence; admittedly some tend to appear early in the period of hypoglycæmia and some tend to appear late, but mostly their appearance is no indication of the stage of hypoglycæmia which has been reached.

The best order in which to discuss them would appear to be according to their severity or seriousness, so that those appearing early on the list may be regarded as normal accompaniments of the hypoglycæmic state and those appearing later may be regarded as more serious and possibly an indication for terminating the hypoglycæmia.

1. Usually the first sign of hypoglycæmia is sweating, and this of course is to be regarded as a normal state. It usually begins during the first or second hour of hypoglycæmia, and as the coma is approached, becomes profuse. Frequently at the end of the day's treatment the patient's clothes and the bed clothes are simply saturated with sweat, and the patient may lose up to two pounds in weight during the morning.

2. Drowsiness usually appears during the second hour and becomes more pronounced until coma supervenes.

3. Restlessness is common and usually appears during the second or third hour. It is usually of moderate degree and subsides as the coma approaches. Sometimes the restlessness is severe and may require two or three nurses to control it. Occasionally the restlessness approaches a state of furor; the patient shouts out, violently attempts to get out of bed and beats the air wildly with arms and legs. In such cases some form of mechanical restraint is essential, and the best plan seems to be to pass a wide strap, such as is sometimes used on anæsthetic tables, around the bed and over the patient's chest, the latter being protected with pillows. This prevents the patient from sitting up, and if the strap includes the arms two or three nurses can safely control the head and legs. The worst degrees of restlessness, however, are an indication for termination of the hypoglycæmic state, since both patient and nurses rapidly become exhausted. The intramuscular route is the best way of giving the glucose, as the restlessness makes other methods very difficult. Future excitement can usually be avoided by manipulation of the dosage of insulin. The dose that produced restlessness is reduced to 10 or 20 units for two days, and then suddenly increased to slightly more than the dose that caused the restlessness. This method, combined with a dose of one or two drachms of paraldehyde shortly before the excitement is expected, is usually effective.

4. An interesting phenomenon observed during hypoglycæmia is *Schnauzkrampf* or "pig's snout". This consists in the pursing of the muscles of the mouth until the mouth assumes the shape of a pig's snout. It is a condition commonly seen in schizophrenia, particularly in cases of catatonia, and it is an interesting fact that it is often reproduced during hypoglycæmia.

5. Profuse salivation is one of the commonest signs seen in hypoglycæmia, and during coma may assume such proportions that a constant stream of saliva flows to the bed and thence to the floor—the so-called "rope" of saliva. To obviate the risk of inhalation of this saliva the comatose patient is usually propped up in full Fowler's position with the head bent forward.

6. Exophthalmos of a fairly pronounced degree is sometimes seen; it normally occurs in the precomatose state, but sometimes is associated with a fairly rare condition in which the eyes remain wide open during coma, the so-called "vigilant coma".

7. Various muscular spasms occur during insulin treatment, and these frequently affect the muscles of mastication; as a result the jaws clamp together with surprising power and tongue biting is a very common difficulty. It is wise to have a suitable gag on every bed,

and sometimes the patient passes the entire period of coma clenching a gag between the teeth.

8. Clonic muscular spasms appear at some time of the treatment in a majority of cases. The mildest form of this myoclonia affects the muscles of the face, particularly those of the mouth and eyes; the twitches occur every few seconds and may last for some time; they usually precede the onset of coma. This condition may subside and give place to the coma or the facial twitchings may be followed by more vigorous generalized myoclonic jerks.

9. This generalized myoclonia may follow on the facial twitchings, as just described, or may arise independently. It consists of a series of very sudden clonic jerks affecting the entire body, particularly the limbs; it can be of all grades of severity, and in its most severe form the limbs may jerk several inches off the bed and the whole bed shakes. The jerks commonly appear at intervals of two to five seconds and the condition may persist for as long as half an hour. They usually occur in the precomatose state, but sometimes quite severe generalized myoclonia will occur so early in the hypoglycæmic period that the patient is sufficiently awake to be aware of them, and afterwards complains bitterly of this unpleasant experience. Generalized myoclonia usually subsides as coma approaches, but not infrequently the condition becomes increasingly violent and passes suddenly into a major epileptiform seizure.

10. The hypoglycæmic seizure appears to be identical with that seen in epilepsy or during "Cardiazol" treatment. It is commonly preceded by generalized myoclonic jerks, and with experience it is possible to know when the seizure is likely to occur and to be prepared with the gag. Sometimes, however, the seizure appears without warning. It passes through the usual tonic and clonic phases, and just before the big inspiratory effort which follows the period of apnoea the patient's colour may be truly alarming, commonly being extremely cyanotic, sometimes of a death-like pallor. These seizures are of frequent occurrence during hypoglycæmia and usually occur during the precomatose state; they may occur, however, at almost any time in the period of hypoglycæmia. The earliest seizure I have have seen occurred one and a half hours after the injection of insulin, the latest about ten minutes after the patient had been fed with glucose. I have also seen a seizure occur at the end of the intravenous administration of glucose.

Many patients go through a long course of insulin treatment without having a seizure; others seem to have a predilection for these seizures and may have as many as eight or more during treatment. The seizure was formerly regarded as an undesirable and dangerous complication; nowadays its therapeutic value has been recognized and it is generally considered perfectly safe.

I think that there is some danger in treating the fit too lightly, particularly if it occurs during the first stage of insulin treatment—that is, before the coma dose has been reached. Only two patients have died at Mont Park as a result of insulin treatment, but both had seizures very early in the course of treatment.

There are several ways of dealing with the hypoglycæmic seizure. (i) After the seizure the blood sugar level rises, and provided the hypoglycæmia has not been too deep, this results in the patient's awakening sufficiently to take a glucose drink within ten or fifteen minutes. (ii) If the seizure does not counteract the hypoglycæmia sufficiently to enable the patient to drink, the coma may be allowed to continue and then terminated as usual by nasal feeding. (iii) Probably the safest method of dealing with a seizure, particularly a first seizure occurring early in the course of treatment, is to give glucose immediately by the intravenous route.

It is interesting to note that the increase in blood sugar content resulting from the seizure lasts only for half to two hours, and so when the patient is roused by the fit and takes the glucose drink he should be given some additional carbohydrate within half an hour, otherwise he may lapse back into coma. The seizure seems to have a most disorganizing effect on carbohydrate metabolism, and it is common for patients who have had a seizure to return to a comatose state even in the afternoon after a normal meal.

Like the epileptic seizure, the hypoglycæmic seizure can be very effectively controlled with "Luminal" when it is considered undesirable or dangerous for a patient to have a seizure, and, as in epilepsy, the sudden cessation of the administration of "Luminal" will increase the liability to seizures. This fact can occasionally be used to therapeutic advantage; for example, it is often desirable to induce seizures during the hypoglycæmic state, and this is done by the intravenous administration of "Cardiazol". If the veins are very small, however, it is desirable to preserve them for the giving of glucose during possible hypoglycæmic emergencies, and in such cases, if the patient is given "Luminal" throughout the first stage of the treatment and the administration of "Luminal" is then suddenly stopped, the patient will usually have one or two seizures during the following week's treatment.

Before I leave the subject of hypoglycæmic seizures it should be mentioned that Sakel originally described two types of coma, the wet and the dry. In the wet type of shock sweating is profuse and seizures do not occur; in the dry type there is little or no sweating and seizures are very common. In my experience it is not possible to draw such a hard and fast line.

11. Another interesting condition sometimes seen during hypoglycæmia is a fine generalized tremor, usually associated with a moderate degree of tonic extension of the muscles, and very similar in appearance to an "æther tremor". It is of no practical importance, and always seems to occur after the tube feeding when the patient is waking from the coma.

12. The so-called tonic extensor spasms are a common accompaniment of hypoglycæmia. During these tonic spasms or tonic stretchings the arms are extended, the wrists flexed and the forearms hyperpronated. The legs are extended and the feet are in the position of extreme equinus. These spasms commence quite abruptly and last for any period up to two minutes; they break down with equal suddenness, and the musculature resumes its usual flaccid state. Usually they recur every few minutes and sometimes there is almost a "trigger-action", the slightest interference with the patient tending to bring on a spasm. These tonic spasms in their mildest form usually occur alone, but when severe they are always accompanied by various respiratory disturbances, particularly the so-called "asthma" type of breathing.

13. The combination of tonic stretching and asthma breathing is a common and alarming syndrome. The patient suddenly passes into a severe tonic stretch, with arms and legs extended, muscles tense, and considerable hyperextension of the spine. Associated with this is a severe expiratory dyspnœa, inspiration being unimpeded, but expiration being very difficult, presumably owing to spasm of the laryngeal muscles. The dyspnœa becomes intense, the patient becomes very cyanosed, and the forcible attempts at expiration produce considerable bulging in the region of the anterior triangles of the neck. Associated with this severe tonic stretching and asthma breathing are a very rapid pulse and dilated, fixed pupils. These severe spasms, like the simple tonic spasms, come in cycles, each spasm lasting two or three minutes with a variable interval between, and like the simple tonic spasms they may be started by any interference with the patient. As the condition progresses the patient assumes a position of opisthotonos, and this, associated with the dyspnœa, cyanosis and rapid pulse, presents a very alarming picture indeed. All attempts at relief, such as posturing, the administration of "Carbogen" *et cetera*, are unavailing; but although the condition looks such a desperate one, it is in reality fairly safe and may be permitted to continue for about half an hour without danger. If it should persist beyond this time, interruption of the hypoglycæmic state by the intravenous administration of glucose is probably wise.

14. Occasionally during deep coma the patient will quite suddenly regain consciousness. This spontaneous termination of the coma occurs in about 2% of cases, and it is always preceded by the syndrome of severe tonic stretching

with the asthma breathing which has just been described. Most patients who present this spontaneous termination of the coma do so on only two or three occasions throughout the course of treatment; but one girl treated at Mont Park awakened spontaneously almost every day.

This patient used to go into a deep coma during the third hour of hypoglycæmia, and soon afterwards the syndrome of tonic stretching and asthma breathing would develop; after about a quarter of an hour of this she would suddenly awake from the coma and demand a glucose drink; if this was refused she would lapse back into coma again within about ten minutes.

The obvious explanation of this phenomenon would be a sudden discharge of adrenalin, with mobilization of the liver glycogen. It should be remembered, however, that it is difficult to terminate coma by injecting adrenalin, and since these spontaneous awakenings are always preceded by muscular spasms, and since a seizure also appreciably raises the blood sugar level, it is probable that the phenomenon is due to the liberation of glycogen following the muscular activity.

15. Other serious disturbances of respiration occasionally occur. A type of tidal breathing, similar to Cheyne-Stokes respiration, may occur; the respirations become deeper and more stertorous, and then subside, to be followed immediately by another *crescendo* without an intervening period of apnœa. This condition is not common and does not look particularly dangerous; but since it occurs only in deep coma, it is probably wiser to terminate the condition, or at least to give a small quantity of glucose to lighten the shock. Typical Cheyne-Stokes breathing has been described, but I have never seen it.

A more serious respiratory disturbance, which appears to follow the pseudo-Cheyne-Stokes breathing, is a slow, shallow type of respiration, combined with extreme pallor and dilated, fixed pupils. This condition seems very similar to that seen during dangerously deep general anæsthesia, and should be regarded as an indication for immediate termination of the coma by the intravenous administration of glucose.

It is not easy to draw a hard and fast line between those hypoglycæmic states which should be regarded as normal, and those which should be regarded as complications. Of the conditions so far described, sweating, salivation and restlessness are of course normal; the myoclonia tonic extensor spasms and seizures, although usually indicative of deeper hypoglycæmia, rarely cause anxiety, and should also be regarded as normal. The Cheyne-Stokes type of respiration and the slow, shallow breathing, however, seem to indicate dangerously deep hypoglycæmia, and should be regarded as complications.

There are two other conditions in which the coma should be terminated immediately; these are laryngeal spasm and cardiac complications. Laryngeal spasm, in which stridor develops and the patient becomes cyanosed, is not common, and in the only two cases in which I have seen it, it has been mild. Attempts to pass the nasal tube may provoke a more severe spasm, and so the coma should be terminated by the intravenous administration of glucose.

Cardiac complications are not nearly so common as one would expect. A pulse rate below 60 or above 120 per minute is commonly regarded as an indication for termination of the coma, but a certain amount of liberty may be taken with these figures, provided the patient's condition is otherwise satisfactory. During the tonic extensor spasms the pulse rate may become almost uncountable, but this is of little consequence, providing that it returns to a reasonable rate between the spasms. A persistently rapid, thready pulse between the spasms is an indication for immediate termination of the coma. Harmless irregularities of the pulse, such as extrasystoles and dropped beats, are common and should not cause anxiety.

Acute pulmonary œdema has been frequently described; but it should be remembered that many patients during insulin coma, in spite of all precautions, aspirate quite a considerable quantity of saliva, and that the bronchial secretions may be profuse, so that the patients become very "bubbly" in the chest. If in addition some respiratory distress, cyanosis and tachycardia are present, the picture is very similar to that of acute pulmonary œdema. However, some glucose given by the intravenous route and a few coughs quickly relieve the condition, which is due to the lungs' becoming water-logged through faulty posture rather than to genuine pulmonary œdema. However, the condition can undoubtedly occur, and at Mont Park there have been two cases, one of which was due to an unfortunate accident rather than to any cardiac involvement.

The patient was in a fairly deep coma, and a nasal tube was passed in order to give glucose. The position of the tube in the stomach was not verified, however, and after several ounces of 50% glucose solution had been poured down the funnel, the patient became extremely cyanosed. The tube was immediately withdrawn, some glucose was given intravenously, and the patient awoke coughing and spluttering. He seemed fairly well for about an hour, and actually had some lunch; then suddenly acute pulmonary œdema developed, and during that afternoon and evening he coughed up twelve pints of frothy blood-stained fluid. An empyema and probably a pulmonary abscess developed; but, thanks to a robust constitution, he eventually made a complete recovery.

This case illustrates the importance of proving that the tube has entered the œsophagus before administering glucose.

Complications Occurring Apart from the Coma.

There are some complications of insulin treatment which occur apart from the coma.

1. Boils are particularly common, but usually they are not an indication for interruption of the treatment.

2. An evening temperature in the region of 100° F. is common, and its only importance lies in the fact that if too much attention is paid to it, it may be allowed to interfere with the course of treatment. Theoretically, this temperature may be due to some developing intercurrent infection; but in practice it seems always to be due to the effects of insulin, and unless some obvious cause is found, I think it should be ignored, as otherwise the patient will receive an interrupted and most unsatisfactory course of insulin treatment.

3. Pneumonia, as one would expect, is a likely complication of insulin treatment, although at Mont Park no case has occurred. Profuse sweating and low temperatures occur during the coma, and in addition coarse bubbling râles may often be heard at the bases of the lungs, so it is rather surprising that pneumonia does not occur more often. Careful nursing would appear to be an important factor in prevention. With regard to low temperatures during the coma, it is worth noting that readings as low as 86° F. have been recorded.

4. An important complication is that known as after-shock. In this condition the patient recovers normally from the morning coma, has a good midday meal, and then some time later symptoms of hypoglycæmia recur; if untreated, the patient relapses into coma. After-shock often occurs when the morning's treatment has resulted in a seizure, and not infrequently the condition is ushered in with a seizure. The latest after-shock I have seen occurred at 5 a.m. on the morning following the coma—that is, twenty-two hours after the injection of insulin—and this in spite of two normal meals in the interim.

Usually the condition occurs during the afternoon, and is easily treated, by a glucose drink if it is recognized in time, by the intravenous administration of glucose if the patient is unable to swallow. When it occurs during the night it is a real danger, because the comatose state may be mistaken for sleep, and if it is left untreated, it may develop into an irreversible coma, a condition which will be described later.

Because of the risk of after-shock, patients undergoing insulin treatment should always be under observation during the afternoon and night, and no patient should be discharged from hospital on the same day that insulin treatment has been given.

Before I leave the subject of the manifestations and complications of hypoglycæmia, it is worthy of note that each patient seems to have a partiality for a particular type of hypoglycæmic reaction. For example, if the coma is quiet

and peaceful on the first few days, this type of reaction will persist throughout treatment. If a seizure or an after-shock occurs early in treatment, these conditions are likely to recur from time to time. If the coma on the first day is of the severe tonic stretching variety, this condition will persist throughout treatment. Similarly, once a patient has exhibited such conditions as myoclonia, exophthalmos or "pig's snout", the same type of reaction will almost certainly recur during coma on future days.

Protracted or Irreversible Coma.

I now come to the discussion of one of the most interesting and most dangerous conditions seen during insulin treatment—that is, the protracted or irreversible coma. The protracted or irreversible coma is really a complication of insulin treatment; but it is of such importance that I think it should be considered separately.

We have seen that Sakel's treatment consists in the giving to the patient of a dose of insulin sufficient to produce a moderate degree of coma, and then in the termination of the coma after a suitable interval by giving glucose through the nasal tube. Normally the patient awakes from the coma within ten minutes to half an hour of the administration of glucose; but not infrequently the patient fails to awake, and is still deep in coma half an hour later. In such a case glucose is given by the intravenous route, and usually the patient awakes immediately; occasionally, however, the coma grows deeper and the patient's condition becomes alarming. In other words, the coma has become irreversible or protracted, and instead of being in a position to terminate the coma at will, the physician has lost control.

The first two to four hours of the irreversible coma may be regarded as the dangerous period. There are often very severe spasms of tonic stretching and asthma breathing, so that the body is in a rigid state of opisthotonos, often literally resting on the heels and occiput, and the respirations are so laboured and the patient is so cyanotic that death from asphyxia seems imminent. This state may be replaced by one of wild restlessness, in which the unconscious patient thrashes about in the bed and rapidly becomes exhausted. Sometimes there is a series of seizures, which differ from the usual hypoglycæmic seizure in that they are at first one-sided and have no tonic phase; they are not so severe as the usual seizure, but in view of the patient's serious condition they are sufficiently alarming. Another condition commonly seen in the early stages of the irreversible coma is great restlessness combined with most intense air-hunger. Accompanying these serious manifestations of the irreversible coma, severe hyperpyrexia is always present, a temperature up to 105° F. being common, and the pulse is rapid and thready, so that the patient's condition appears desperate.

After this dangerous period of two to four hours, the patient lapses into a quiet, deep coma, which may last for two, three or four or more days, but which, owing to the relatively reassuring appearance of the patient, may be regarded as the safe period. During this quiet comatose state artificial feeding must be commenced, and after a few days the condition passes into one of deep somnolence during which the patient can be roused just sufficiently to take fluids by mouth. This intense drowsiness gradually wears off, and at the end of perhaps a week it is possible to make some examination of the mental state. This is always of the organic reaction type, the memory being grossly impaired and disorientation pronounced. With a very few exceptions this mental clouding clears up in a period of one to six weeks, and frequently one finds a dramatic improvement in the psychotic condition from which the patient originally suffered.

The Treatment of Irreversible Coma.

Irreversible coma is not recognized until, the tube feed having failed to arouse the patient, glucose is given intravenously and immediately precipitates alarming symptoms. Therefore the first point to emphasize is that, although dangerous symptoms of what we call hypoglycæmia are present, the patient is actually hyperglycæmic, and consequently to give further sugar is useless. It is wise, however, to give a little glucose intramuscularly to form a depot and so ensure that the blood sugar will not sink below normal levels.

Having given sufficient sugar, all one can do is to try to treat the alarming symptoms of the danger period, and for this a host of remedies have been tried. Vitamin B_1 given intravenously has not the slightest effect on the condition, although if insulin treatment is resumed later, its use prophylactically does seem to lessen the severity of a second irreversible coma. Calcium has been used intravenously in an attempt to control the tonic spasms of muscles; it appears to me to be quite useless. Adrenalin has been recommended, but its effect of raising the blood pressure is dangerous and may provoke seizures, and its use should be abandoned. The administration of "Carbogen" is rational treatment when air hunger or asthma breathing is present, and on occasions it seems to help. Lumbar puncture may be employed, but its value is, I think, very doubtful. However, I have seen a patient in a state of severe irreversible coma with repeated seizures stop having seizures after lumbar puncture, although the cerebro-spinal fluid appeared to be under normal pressure. When there is a risk of cardiac or respiratory failure, and there frequently is such a risk, "Coramine" given intravenously seems to do good.

More insulin has been given in an attempt to combat the irreversible coma. It should be remembered, however, that no ketosis is present and that the hyperglycæmia can hardly be contributing to the unconscious state, and consequently, as one would expect, insulin does not affect the condition.

Blood transfusion has been used overseas.

For patients who have repeated seizures or wild thrashing restlessness, there is one remedy which is undoubtedly of great value, and that is light chloroform anæsthesia. This may seem a dangerous procedure, particularly when large doses of morphine and barbiturates have previously been given in an attempt to stop the seizures or the restlessness; but it must be remembered that the patient's condition is already desperate, and repeated seizures will almost certainly end in exhaustion unless they are controlled. In my experience they can be controlled, effectively and safely, with a few inhalations of chloroform.

Apart from the application of the few symptomatic remedies that have been mentioned, there is nothing one can do during the dangerous period of the protracted coma.

At the end of this dangerous period, a quiet, deep coma follows, and during this phase the pulse, colour and respirations are all satisfactory and one feels that it is only a matter of time before complete recovery takes place. During this period, which may last two, three or four or more days, the important thing is to supply sufficient fluid. If a nasal tube is passed at the beginning of this period—that is, on the afternoon of the first day of the protracted coma—the entire glucose feed that was given at midday to terminate the coma can often be asppirated from the stomach. This gastric retention is a possible ætiological factor in the protracted coma, and when it is present one should never depend on the nasal tube for supplying fluid during the early comatose period.

Usually the patient, although relatively quiet, is still too restless for the continuous intravenous administration of saline solution, but often saline solution with 5% glucose added may be given by the continuous intramuscular method. Failing this, saline solution must be given by the rectum, and small quantities of glucose should be administered from time to time, either intravenously or intramuscularly. It is desirable to give just enough glucose to maintain a mild glycosuria. On the second and subsequent days nasal tube feeding may be employed with safety, as the gastric retention will certainly have passed off. This is all that is necessary during this quiet comatose period, and at the end of a few days the patient can usually manage to take fluids by mouth.

The cause of protracted coma is quite unknown. In the United States of America, 2% of patients receiving insulin treatment have protracted coma. At Mont Park the incidence is over 10%. A possible reason for the much greater incidence at Mont Park would be the use of larger doses of insulin; but the average coma dose for males at

Mont Park is 170 units, which is almost identical with that used in other clinics with a much lower incidence of protracted coma. Another factor of which one immediately thinks is the period for which the patient is left in coma; but again, the periods of coma at Mont Park are no longer than those employed in the United States.

The phenomenon of gastric retention suggests that an unduly long period of hypoglycæmia is of some importance. In these cases the blood sugar level is not raised until glucose is given intravenously about half an hour after the nasal feed, and it seems possible that this delay of half an hour renders the patient unable to utilize the available sugar. However, in certain modifications of insulin treatment the period of hypoglycæmia is extended to twelve hours, and I have not heard of protracted coma occurring under these conditions.

Undoubtedly the insulin-tolerant patient is more prone to protracted coma. It has been seen that most patients during insulin treatment can have their original coma dose progressively reduced—that is, they are insulin-sensitive. Sometimes, however, the dose has to be constantly increased in order to obtain a coma—that is, the patient is insulin-tolerant; it is these patients who in my experience develop irreversible coma.

It appears likely that the cause of protracted coma lies in the patient rather than in the method of giving insulin treatment. Some patients seem to be able to stand prolonged hypoglycæmia and to recover immediately glucose is given; in other patients the reversible changes in the nerve cells which accompany the normal coma become irreversible after a relatively brief period of hypoglycæmia.

To me, the great mystery of protracted coma lies in the fact that it is only when the blood sugar is restored to normal levels that alarming symptoms appear. When the patient fails to arouse himself after the routine nasal feed, his condition is not unduly alarming. At this stage, owing to the gastric retention of the glucose, he is probably still hypoglycæmic; as soon as the glucose is given intravenously, however, the serious symptoms develop. In other words, the restoration of the blood sugar to an approximately normal level definitely causes the condition to become worse. It almost appears as if the patient were sensitive to glucose, and consequently it may be better to desensitize him with small repeated intramuscular injections, rather than to give a large quantity into a vein.

One cannot help wondering what would happen if no glucose were given intravenously and the patient allowed to remain hypoglycæmic, on the assumption that the gastric retention is an attempt on the part of nature to prevent glucose from reaching the blood stream. I am well aware that such a suggestion must sound ridiculous; but one cannot escape the fact that it is the restoration of the blood sugar to normal levels that precipitates the serious symptoms in this condition.

Points of Interest to General Medicine.

Tonight we are concerned mainly with psychiatric matters; but insulin treatment has brought to light certain facts, which are of such interest to general medicine that I feel they should be emphasized.

The first of these is the surprisingly large doses of insulin that the human organism can tolerate. Before the advent of this treatment, most of us, giving evidence on oath, would have stated unequivocally that a fasting patient who was given 600 units of insulin would certainly die. We now know that such a dose can be repeated day after day and can result in an improvement in the general physical condition.

The second point of interest is the administration of glucose by the intramuscular route. Glucose is commonly held to be irritating to the tissues and to lead to necrosis and sloughing, and so when given post-operatively it is always by the intravenous route. It can be definitely stated, however, that glucose given intramuscularly is perfectly safe and harmless. At Mont Park, glucose solution has been given intramuscularly on about 300 occasions and I have not once seen the slightest harm result. The practice is to give 30 cubic centimetres of 15% aqueous solution into the lateral aspect of each thigh, and since this method does no harm, the slower administration of 5% glucose in saline solution by the continuous intramuscular drip method must surely be equally safe.

Until fairly recently, there was a considerable difference of opinion as to whether glucose was absorbed when given by the bowel. In my experience it is quite impossible to terminate a hypoglycæmic coma by glucose given rectally, and furthermore, glucose administered rectally soon after a coma dose of insulin does not seem to affect either the time of onset or the depth of the ensuing coma.

Another point that I feel is of general interest is the small amount of glucose which is sufficient to terminate a hypoglycæmic coma. Provided the coma is still reversible, 20 grammes of glucose given intravenously will always terminate it, and this amount is quite independent of the dose of insulin which produced the coma. There is really nothing surprising in this fact when we remember that 20 grammes of glucose will raise the blood sugar level from zero to 0·35%; but it seems to me that it is often overlooked in the treatment of a diabetic patient who has been taking insulin and has developed a hypoglycæmic coma. If such a patient is given 20 grammes of glucose intravenously and fails to awake, it seems likely that the coma has become temporarily irreversible, in which case the immediate administration of more glucose is quite useless.

Lastly, it may seem surprising that in this discussion of deep hypoglycæmic states and irreversible coma, no mention has been made of blood sugar estimations. Blood sugar readings are of considerable interest, but they are of little practical value in an insulin clinic. The quickest method of making estimations at the bedside takes approximately fifteen minutes, and furthermore the blood sugar level gives no indication of the danger or depth of the coma, so that the patient's condition must always be assessed on clinical grounds and not on laboratory findings. Extensive research has been done overseas on the blood sugar level during the hypoglycæmic coma, and the results are so surprising that I feel they should be more generally known. During the hypoglycæmic period, blood sugar readings of 0·04% and 0·02% are common, and on occasions the blood sugar level may fall to almost zero. These readings, however, give little indication of the clinical condition of the patient. When the fasting patient is given his coma dose of insulin, the blood sugar level is, of course, about 0·09%. The maximum fall occurs during the first hour or hour and a half; but at this stage the patient is still wide awake and showing only slight signs of the hypoglycæmia, although the blood sugar level may be almost zero. From this point the blood sugar level usually steadily rises, and at the beginning of the third hour is frequently double the earlier minimum reading; at this stage the patient begins to go into coma. So we are faced with the surprising fact that signs of hypoglycæmia and coma appear only when the blood sugar level begins to rise.

Dangers of Insulin Treatment.

The dangers of insulin treatment are best examplified by the two deaths which have occurred as a result of insulin treatment at Mont Park—a mortality rate of less than 1%.

The first patient who died was a man, aged forty-seven years, who had suffered from paraphrenia for several years. No abnormality was found on physical examination, except some mild peripheral vascular sclerosis. The systolic blood pressure was 155 millimetres of mercury and the diastolic pressure 95; the exercise tolerance was good.

On the first day of treatment 20 units of insulin were given with no effect. On the second day 40 units produced no change. On the third day 50 units were given, and at midday he was still wide awake and showed no signs of hypoglycæmia. He refused to drink glucose and was stood up beside his bed; immediately he had an atypical seizure, consisting of clonic spasms of the right leg, tonic spasms of the right arm and deviation of the head to the right. Within a minute of the onset of the seizure he became cyanosed, the veins of the neck became dilated, and the heart failed. Respiration continued for a minute or two after the heart had apparently stopped. At the post-mortem examination intense congestion of all the internal organs was found and the lumen of the left coronary artery was almost occluded.

The cause of death in this case was obviously unrecognized coronary artery disease, and one may

question the wisdom of giving insulin treatment to a patient aged forty-seven years. However, since this accident patients considerably older than this have been successfully treated at the hospital, and several of them have had the combined insulin and "Cardiazol" treatment, with complete success. Possibly the taking of an electrocardiogram as a routine measure in these older patients would be wise. An interesting feature of this case is the occurrence of a seizure as the first sign of hypoglycæmia.

The second death was that of a girl, aged eighteen years, suffering from schizophrenia, possibly on a basis of mild congenital mental deficiency. During the first ten days of insulin treatment the dose was regularly increased to 200 units, and during this period she showed no signs of deep hypoglycæmia and was able to take her glucose drink each day. On the eleventh day 220 units were given, and again at midday the patient was able to take her glucose drink.

On the twelfth day 240 units were given, and early in the third hour of hypoglycæmia severe generalized myoclonia developed and eventually terminated in a seizure. She awoke spontaneously after the seizure and was able to take a glucose drink. At 1 p.m. she vomited; at 2 p.m. she felt nauseated and refused lunch, but was wide awake. At 2.30 p.m. she became drowsy, had a second seizure, and was given glucose by the intravenous and intramuscular routes. At 3.30 p.m. she was still very drowsy and had a third seizure. That afternoon and evening she remained in a lightly comatose state and was given glucose and saline solution by the intramuscular route.

The next morning she was in a quiet deep coma and her condition seemed fairly satisfactory; but during the afternoon the temperature rose to 105° F., the pulse rate was 120 per minute and the respiratory rate was 50 per minute. Her condition became progressively worse and she died early the next morning of respiratory and cardiac failure. The post-mortem examination revealed slight congestion of the bases of the lungs; otherwise no abnormality was found.

The important points in this case are, firstly, that the earliest sign of deep hypoglycæmia—that is, the seizure—appeared before the coma dose had been reached; and secondly, the patient awoke after the fit, but an after-shock occurred which in turn developed into an irreversible coma. In a review of the treatment it will be remembered that after the seizure the patient was allowed to awake spontaneously and take a glucose drink; it would probably have been better to give glucose intravenously at this stage. Furthermore, much of the glucose that was given by mouth was vomited and lunch was refused on account of nausea. At this stage I think glucose should certainly have been given by the intravenous route, and I believe that it might have prevented the fatality. Once the coma had become irreversible there was little one could do.

This case illustrates, firstly, the danger of a first seizure occurring early in the course of treatment, and secondly, the liability to after-shock of patients who have had seizures.

Psychotherapy.

It should be emphasized that simple, common-sense psychotherapy is an essential part of insulin treatment. This matter will be discussed more fully when "Cardiazol" treatment is dealt with; and for the present it must suffice to state that, unless the recovering patient is given some psychological guidance, he will not achieve as complete a remission as would otherwise be possible.

The Results of Insulin Treatment.

It is convenient to discuss the results of insulin treatment in conjunction with the results of "Cardiazol" treatment, and so these matters will be considered together in the next lecture.

EXOPHTHALMOS IN GOITRE.

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Exophthalmos has always been regarded as an inconstant, although important, feature of thyreotoxicosis. Parry, Graves and Basedow all recognized it as a very evident clinical feature. Exophthalmos is sometimes the earliest sign of the disease, and when treatment is given it frequently persists long after the other signs and symptoms have disappeared. Graves wrote as follows of one of his patients:

> The eyes assumed a singular appearance, for the eyeballs were apparently enlarged so that when she slept the lids were incapable of closing. When the eyes were opened, the white of the sclerotics could be seen, to a breadth of several lines, all round the cornea.

Despite this early recognition and the fervid attempts of anatomists, physiologists, pathologists and surgeons to elucidate the pathogenesis of exophthalmos, the mechanism of its production is not well understood. The voluminous literature of the subject is frequently confused by writers who fail in their discussions to differentiate between the production of hormones or toxins at a distance from the orbit, and the local mechanism responsible for the exophthalmos.

The Diagnosis of Lid Retraction and Exophthalmos.

On the clinical side little attempt has been made to distinguish between true exophthalmos, on the one hand, and apparent exophthalmos, which is caused by widening of the palpebral fissure. Retraction of the upper eyelid is frequently responsible for the latter. Both lid retraction and exophthalmos may of course be associated, but frequently the former is present alone. Some illustrations of unilateral exophthalmos reveal undoubted spasm of the upper eyelid without the presence of proptosis. In general, too, exophthalmos has been regarded as a more or less evident clinical sign, the presence of which may be confirmed by a number of standard tests. Often the protrusion can be determined only by instrumental measurement. Perhaps the great variation in prominence in the eyes of normal people, and the characteristic appearance of many patients in the early stages of exophthalmic goitre, have prevented the more general use of an exophthalmometer. Moreover, increase or decrease in the degree of proptosis, from the previously normal degree for that patient, is of greater significance to the clinician than is the measured position of the cornea relative to the lower or external orbital margin. In view of these facts, all statistics giving the frequency of exophthalmos in different types of toxic goitre and during different age periods, are unreliable. There is no doubt, however, that both retraction of the upper eyelid and true exophthalmos are much more frequent in patients having thyreotoxicosis associated with diffuse thyreoid hyperplasia than in those who have nodular toxic goitres.

Experimental work directed toward the solution of the problem has yielded most interesting data, but owing to variations in the anatomy of the orbits of different animals, conflicting results are so frequently found that great caution is necessary in making comparisons with human exophthalmos.

In recent years certain writers (Friedgood, Naffziger, Stallard, Brain) have discussed as a separate entity cases of extreme exophthalmos, particularly if they are associated with post-operative hypothyreoidism or with ocular palsies. While in some instances one finds it impossible to know whether a particular case should be placed in this category, an attempt should be made to distinguish these severe and progressive cases, in which destruction of the globe ultimately occurs, from the more usual lesser degrees of proptosis.

The literature abounds with descriptions of eye signs which are characteristic of exophthalmos, and Joll publishes a long list of them. It may be repeated, however, that the absolute degree of exophthalmos can be accurately measured only by an exophthalmometer, and that many of the classical "signs" are indicative of spasm of the non-striated muscular portion of the *levator palpebræ superioris*, rather than of proptosis. Because of this only a few of the more commonly used signs will be considered. Dunhill finds that in over 80% of cases of primary toxic goitre von Graefe's sign is present. The upper lid lags behind the eyeball as the patient slowly looks downward from the horizontal, the movement of the lid being jerky rather than evenly graded. This sign, like that of

Dalrymple—namely, widening of the papebral fissure—is due to spasm of the upper lid rather than to exophthalmos; it is occasionally found in a wide variety of pathological states, many of which are characterized by hypertonus of the sympathetic nervous system—for example, hypertension, chronic nephritis and pulmonary tuberculosis. Infrequency of winking (Stellwag) is probably of similar significance, but weakness of convergence (Mobius) is in most cases due to true exophthalmos. Failure to wrinkle the forehead on looking upward ("Joffroy's sign") is observed so frequently in normal people that it is of no value in diagnosis.

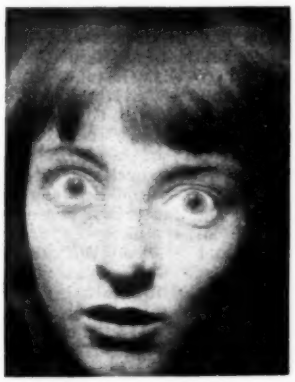

Figure I.
Photograph showing the combination of lid retraction and exophthalmos.

The features of lid retraction may be compared and contrasted with those of exophthalmos. In lid retraction the upper lid is raised so that the free border is elevated, and when the eye looks forward a band of sclera may be seen above the cornea. The lower lid often appears to be raised a little, and it covers the lower margin of the cornea so that no sclerotic can be seen below it. In proptosis a band of sclerotic is seen below the cornea. In cases of unilateral lid retraction these features are most

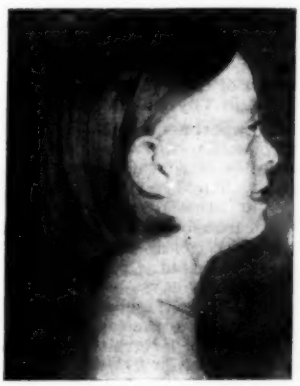

Figure II.
Photograph of a child operated on for toxic goitre. Exophthalmos is evident in profile.

conspicuous, and when the eyelids are closed the free border of the upper lid is higher on the affected side, the lower lid being elevated to meet it. The palpebral fissure is therefore straight and high.

A conspicuous fold of skin is seen from six to ten millimetres above the free border of the upper eyelid when the eyes are closed. This fold is sometimes bilateral in normal people.

Pochin noted that in sleep the upper eyelid remained retracted as in light closure, but as the lower lid was not raised to meet it a gap resulted. I have confirmed this observation frequently.

The earliest and most common eye signs in toxic goitre are the characteristic stare, infrequent winking and tremor of the closed lids. Later protrusion of the eyeball occurs. This may be masked if the patient has deeply-set eyes and an overhanging forehead. Weakness of accommodation during convergence is the most common muscle disturbance, and frequently this persists after operation, delaying the patient's return to normal activity. The more serious defects in eye movement are discussed in the section on severe progressive exophthalmos, along with alterations in vision due to papillœdema, optic atrophy and retinal changes. Increased ocular tension is sometimes found, without changes in the fundus characteristic of glaucoma or alteration in the visual fields. Ruedemann encountered eleven cases in a very large series of toxic goitres, and with one exception the eye condition returned to normal after thyreoidectomy. Usually treatment of the toxic goitre greatly reduces or cures the lid retraction and less frequently the proptosis; but when interference with accommodation convergence is pronounced, the improvement in this lesion is least noticeable.

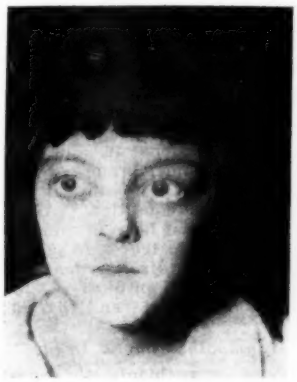

Figure III.
Photograph of a child, showing exophthalmos without lid retraction.

Œdema of the eyelids is an important and frequently overlooked concomitant of exophthalmos—indeed, it should always be sought. Sometimes this œdema completely masks the exophthalmos. I know of a number of instances in which œdema of the eyelids occurred in the early stages of exophthalmic goitre and its significance was for a time overlooked.

Experimental Exophthalmos.

The methods by which exophthalmos has been produced experimentally may be tabulated as follows:

A. Mechanical sympathetic stimulation—
  Brain stem.
  Cervical sympathetic trunk.
B. Sympathetic stimulation by chemical means—
  Sympathomimetic drugs.
  Methyl cyanide.
C. Thyreoid extract—
  Alone.
  Combined with sympathomimetic drugs.
D. Pituitary thyreotropic hormone—
  Alone.
  Combined with androgens.

Exophthalmos has been produced in animals by stimulation of the sympathetic nervous system, directly or indirectly, by the administration of thyroxine or thyreoid extract, and by the injection of pituitary thyreotropic hormone. Probably the earliest attempts to produce exophthalmos by stimulating the nervous system were

made by Filèhne, who in 1879 cauterized the upper portions of the restiform bodies in dogs. Later, Bienfait confirmed his observations by producing similar lesions. Claude Bernard, Langley and Sherrington, and others, found conclusive evidence that the smooth muscle in the orbit was supplied and maintained in a state of tone by the sympathetic nervous system, and they obtained protrusion of the eyes by stimulating the cervical sympathetic trunk.

In 1904 McCallum and Cornell, by stimulating sympathetic fibres in the orbits of dogs, produced waves of contraction in the smooth muscle fibres of the periorbital membrane and showed that the resulting exophthalmos was independent of vascular changes. After division of the periorbital membrane cervical sympathetic stimulation no longer caused exophthalmos. An attempt at more prolonged sympathetic stimulation was made by Cannon, Binger and Fits in 1915. Experimenting with cats, they anastomosed the phrenic nerve to the cervical sympathetic trunk and produced exophthalmos; but subsequent workers have not been able to confirm these results. McCallum and Cornell's work was confirmed and extended by Whitnall and Beattie (1933) and Code and Essex (1935); they showed that in the dog excitation of the periorbital membrane caused the eyeball to be pushed forward. Mechanical stimulation of the human cervical sympathetic trunk does not lead to protrusion of the eyes because there is little smooth muscle in the human periorbital membrane.

The results of animal experiment have been criticized by Essex and Corwen, who found that in most instances the reported exophthalmos was merely that caused by the anæsthetic, and it disappeared when anæsthesia was terminated. Whether this is so or not, the findings have little bearing on the problem of human exophthalmos, for variations in the structure of the mammalian periorbital membrane are such that the results obtained from these animal experiments do not apply to the human condition. Also, in conducting such experiments, one must bear in mind that stimulation of the cervical sympathetic trunk, in addition to affecting the orbital smooth muscle directly, may activate both the thyreoid gland and the pituitary, thus causing an increased secretion of thyreoid and thyreotropic hormone.

Sympathomimetic Drugs.

A wide variety of sympathomimetic drugs has been used in an attempt to produce proptosis both in man and in animals. In normal human beings this is unsuccessful as a rule, but ephedrine hydrochloride will sometimes cause transitory exophthalmos in patients suffering from thyreotoxicosis. In dogs the administration of sympathetic-stimulating drugs, particularly if combined with thyroxine, will cause exophthalmos. Many workers, including Marine and his collaborators, have produced it in rabbits by feeding them on alfalfa hay and oats, or by giving injections of methyl cyanide. The available evidence indicates that cyanides interfere with tissue oxidation, and that as the result of this the sympathetic nervous system is over-stimulated. Any proptosis is abolished by section of the cervical sympathetic trunk.

Kunde first demonstrated that such exophthalmos was more readily produced in thyreoidectomized animals, an observation which has been confirmed by many subsequent observers. Although it has been suggested that the tying of the anterior jugular veins may have been the important factor in its more ready production after thyreoidectomy, more recent work has shown that eye protrusion occurs independently of venous obstruction.

Despite the frequency of exophthalmos in toxic goitre, the administration of thyreoid extract or thyroxine to human beings is rarely followed by protrusion of the eyes. Tachycardia, tremor and a raised basal metabolic rate, on the other hand, can be very rapidly induced. On the basis of this finding many writers have considered exophthalmic goitre to be the result of dysthyreoidism rather than of hyperthyreoidism. Nevertheless, one cannot escape the fact that some patients when given thyreoid extract develop either unilateral or bilateral exophthalmos. Brain and others have reported cases, and I have personally observed the phenomenon. Susceptibility of the individual subject to stimulation by thyreoid hormone must be of great significance.

Thyreotropic Hormone.

Early studies on the effect of thyreotropic hormone extracted from the pituitary gland showed that when injected into animals and birds it was capable of producing exophthalmos. It was soon apparent that, as in earlier experiments with methyl cyanide, a previous thyreoidectomy facilitated the process. Smelser, in a series of well-controlled experiments which were recently confirmed, found that pronounced proptosis could be induced in thyreoidectomized guinea-pigs, even after the cervical sympathetic trunk has been severed. He made careful histological and biochemical studies of the orbital contents, correlating his findings with lesions observed in human "paradoxical exophthalmos". He considers that identical changes are found in the two conditions, and that the human disease results from an excessive output of thyreotropic hormone. In view of their significance, the changes in the orbit will be outlined briefly. The amount of fat is greatly increased, and between the fat-cells lies granular eosin-staining material, presumably of lipoid origin. Collections of lymphocytes are seen scattered throughout the fat and between the muscle fibres of the extrinsic ocular muscles. The ocular muscles are swollen and œdematous, and the individual fibres exhibit toxic spoiling and in many instances loss of their transverse striations. Both the lachrymal glands and the muscles are considerably increased in weight. The greater incidence of experimental exophthalmos in male as compared with female rabbits, and the relative preponderance of males affected with human paradoxical exophthalmos, led Marine to investigate the effect of androgens.

He found that castration caused a gradual recession in the proptosis of animals, while cryptorchids responded normally to the injections. His studies showed also that such androgens as androsterone, testosterone and dehydroandrosterone would not cause protrusion of the eyes if injected alone, but that they increased the exophthalmos produced by thyreotropic hormone.

In short, thyreotropic hormone is the most successful agent known for producing experimental exophthalmos; the proptosis differs from that elicited by other substances in that the protrusion of the eyes is of greater degree, does not disappear after section of the cervical sympathetic trunk or after the death of the animal, and is accompanied by well-defined structural changes in the orbit.

The Mechanism of the Production of Exophthalmos in Thyreotoxicosis.

Although much experimental research has been carried out on the production of exophthalmos in animals, relatively few studies of the human orbit have been made in thyreotoxicosis. The local mechanism responsible for the protrusion of the eyes in severe progressive exophthalmos is reasonably clear; but the method of its production in typical cases of toxic goitre is still the subject of much speculation. The more important of the numerous theories that have been advanced from time to time will be stated first and then critically examined. (i) Exophthalmos is due to the tonic contraction of smooth muscle, either Müller's periorbital muscle, Landström's muscle, or the two combined. (ii) An increase in the intraorbital fat is responsible. (iii) Dilatation of the ophthalmic veins behind the eyeball is the cause. (iv) Dilatation of the arteries posterior to the eyeball is the cause. (v) Exophthalmos is due to œdema of the orbital fat. (vi) An increase in the volume of the extraocular muscles is the cause. (vii) Weakness of the recti muscles associated with other factors tends to cause the eyeball to protrude.

The Smooth Muscle of the Human Orbit and its Relation to Exophthalmos.

In 1858 H. Müller wrote a short acount of the plain muscle found in the orbits of various animals and of man.

He found that mammals other than man possessed a well-defined periorbital membrane (synonym: periorbital muscle) which contained a variable quantity of smooth muscle. This cone-shaped musculo-membranous structure is attached by its apex around the optic foramen and at its base to the orbital margin in front. Some animals by means of this structure are able to protrude the globe, the antagonist to this being the muscle *retractor bulbi*. Human beings have no *retractor bulbi*, and Müller considered that the small greyish-red mass of muscle crossing the infraorbital fissure was the homologue of the mammalian periorbital membrane.

Also in 1859 Müller described the involuntary portion of the *levator palpebræ superioris* muscle and the smooth muscle fibres in the lower eyelid. Much confusion has arisen in discussions on exophthalmos because subsequent writers were ignorant of this, and English-speaking anatomists have not clarified the subject by referring to the smooth muscle of the infraorbital fissure alone as "Müller's muscle". Stimulation of the smooth muscle in the upper eyelid, called Müller's muscle by some Continental writers, will cause retraction of the upper lid and apparent exophthalmos; but it is difficult to see how the contraction of a few muscle fibres bridging the infraorbital fissure (the Müller's muscle of English anatomy) could have any appreciable direct effect on the globe. The smooth muscle of the *levator palpebræ superioris* is supplied by sympathetic nerve fibres, and stimulation of the cervical sympathetic trunk causes retraction of the upper eyelid with widening of the palpebral fissure, and division of the trunk results in ptosis. One may therefore state with certainty that over-stimulation of the sympathetic nervous system can give rise to retraction of the upper eyelid with widening of the palpebral fissure and apparent exophthalmos, but that no muscle described by Müller in the human orbit is able to produce any appreciable degree of true exophthalmos.

William Turner, in 1862, discussing the smooth muscle of the orbit, mentioned that Müller himself recognized that the smooth muscle he described could not account for human exophthalmos.

Landström reinvestigated the smooth muscle of the orbit and published the results of his histological studies in 1907. The contents of several orbits were removed by subperiosteal resection, the lids and *septum orbitale* being undisturbed. After fixation, sections were cut in various planes. He found a cylinder of smooth muscle arising from the *septum orbitale* in front and inserting itself into the eyeball just behind the equator. From the attachments of this smooth muscle sheet, he concluded that when stimulated it was capable of pulling the eyeball forward, the recti acting as antagonists. Subsequent observers have not been able to confirm Landström's findings.

Kraus considered that orbital smooth muscle fibres were by their contraction able to constrict the ophthalmic veins and thus bring about distension of the tributaries in the orbit, so mechanically producing exophthalmos. The matter was reviewed by Hesser, who in 1913, after exhaustive studies, concluded that this was not possible.

Recently the periorbital membrane of both man and animals has been restudied by Russell and Brunton, who describe smooth muscle in the human periorbital fibrous membrane. As previously mentioned, Müller stressed the muscular character of this membrane in mammals other than man, but considered that in the human orbit the only homologous muscle was that rudimentary structure found bridging the infraorbital fissure. These later workers, therefore, differ from Müller in that they describe a more extensive distribution of smooth muscle, and consider that it may be significant in the causation of human exophthalmos. Hesser previously showed a few smooth muscle fibres in drawings from microscopic preparations of the human periorbital fibrous membrane, but regarded them as unimportant. Brunton has published photographs of such fibres lying behind the eyeball in the upper and outer portions of that structure; these are continuous with the fibres crossing the infraorbital fissure, and are found both in normal persons and in those suffering from Graves's disease. They are not found to be larger or more extensive in patients with exophthalmos. After considering the results of Brunton and Russell and studying histological sections through the orbit in full-time fœtuses, I am of the opinion that it is unlikely that the few muscle fibres present could have any noticeable effect on the globe. Experiments on human beings likewise indicate that orbital smooth muscle plays little or no direct rôle in the production of proptosis. Exophthalmos could not be produced by Müller and Wagner, who stimulated the cervical sympathetic trunk in decapitated criminals, or by Unverricht experimenting upon patients undergoing operations on the neck. However, the possibility that long-continued sympathetic hypertonus may produce exophthalmos by some mechanism other than by the contraction of orbital smooth muscle is not denied.

Increased Orbital Fat.

It has been suggested that an increase in the intra-orbital fat causes exophthalmos. In some cases a definite increase in the retrobulbar fat is found, but this is not invariable, and when present may well be the result rather than the cause of the protrusion. On such a basis it is difficult to explain the post-mortem recession of the eyeball noted in many cases of Graves's disease.

Œdema of the Orbital Fat.

Other writers have postulated an œdema of the retrobulbar fat as being significant; but except in rare instances examination of the orbit fails to reveal its presence.

Dilatation of the Ophthalmic Vessels.

Dilatation of the ophthalmic vessels behind the eyeball has been the subject of investigation by Kraus, Hesser and others, who studied the arrangement of the orbital smooth muscle and its relation to the ophthalmic veins. Hesser concluded that, because of its anatomical distribution, muscular contraction could not obstruct the flow of blood in the veins and thereby cause œdema. This does not exclude the possibility of arteriolar and capillary dilatation, which we know occurs elsewhere in the body in thyreotoxicosis. I believe that this is the important factor in the causation of lesser degrees of exophthalmos, and is thyreogenic.

Increase in Volume of the Eye Muscles.

In contrast with the findings in severe progressive exophthalmos, the eye muscles are usually normal both anatomically and histologically. But in some instances there is an increase in their volume, the result of pituitary dysfunction; both the pituitary and the thyreoid glands are therefore responsible for the exophthalmos.

Weakness of the Eye Muscles.

Weakness of the extrinsic eye muscles in itself could not cause exophthalmos; but as the recti, by virtue of their anatomical position, tend to prevent forward displacement of the globe, their lack of tone may facilitate the development of proptosis induced by other agents. However, gross muscular weakness if found in thyreotoxicosis only rarely and therefore cannot be of great significance.

Severe Progressive Exophthalmos.

Severe progressive exophthalmos is also known as progressive exophthalmos (Naffziger), paradoxical exophthalmos (Stallard, Zimmermann), malignant exophthalmos (Friedgood) and exophthalmic ophthalmoplegia (Brain). Exophthalmos is found most frequently in patients suffering from long-standing thyreotoxicosis associated with diffuse hyperplasia of the thyreoid epithelium. In such circumstances gradual improvement in the proptosis usually follows adequate subtotal thyreoidectomy, and if the protrusion of the eyes has not been present for too long a time it may be cured. On rare occasions severe and progressive exophthalmos with defective eye movement

follows treatment which renders the patient otherwise normal or myxœdematous. In addition to such cases of post-operative exophthalmos, many instances are recorded of a similar condition occurring in patients with very mild thyreotoxicosis, or even independently of it.

No entirely satisfactory terminology has been devised for these severe cases of exophthalmos, in which one may observe destruction of the eyes. "Malignant exophthalmos" is a term used by a number of American writers, including Friedgood. This indicates the gravity of the condition; but confusion readily arises between the disease and the exophthalmos associated with malignant tumours of the orbit. "Paradoxical exophthalmos" has been suggested and used by other writers, including Stallard and Zimmermann. The exophthalmos is considered to be paradoxical, for thyreoidectomy makes it more prominent, whereas in the typical exophthalmos of thyreotoxicosis the patient's state is improved by surgical treatment. The term "severe progressive exophthalmos" suggested by myself is probably most suitable, for it is non-committal as to ætiology, and describes the severe and progressive nature of the disease which often continues until the eyes are extruded. It is not wholly satisfactory, for after a high degree of exophthalmos is attained, the eyes may remain unaltered in that position. Should it be proved that the disease is due to an increased production of thyreotropic hormone, the term "thyreotropic exophthalmos" would be desirable. Brain discussed a series of such cases in which the exophthalmos was associated with ocular muscle palsies and suggested that the condition be called "exophthalmic ophthalmoplegia". Some of these cases provided evidence of thyreotoxicosis developing after the exophthalmos; but from a study of the literature one cannot say definitely in what percentage of such cases a toxic goitre appears at some stage of the illness. Friedenwald's case has an important bearing on this problem. Severe exophthalmos developed after infection of a nasal sinus. The patient's condition was at first diagnosed wrongly as a cerebral abscess. Some months later evidence of a toxic goitre developed and the basal metabolic rate was raised to + 60%. Death occurred from thyreotoxicosis, and at autopsy there was no evidence of orbital infection or œdema, but lymphocytes were present in the eye muscles. The pituitary gland was normal histologically. This was undoubtedly a case of severe progressive exophthalmos associated with thyreotoxicosis, of which it was the first sign. Other similar cases have been recorded (Naffziger).

Pathogenesis.

The hypothesis most usually applied to explain the condition is that of an over-production of thyreotropic hormone. The chief evidence for this is that similar orbital changes are found in animals which are injected with that substance (Smelser, Naffziger). At the present time the chief difficulty in accepting this view arises from the fact that an increase in the thyreotropic hormone of the blood or urine has not been demonstrated in patients with toxic goitre, and the pituitary gland has been found to be normal histologically in patients who have died from the disease. It may be that the methods of detecting the hormone are too crude to estimate the slight increase which, acting over a long time, might be responsible for the orbital changes, and that histological normality of the pituitary does not necessarily exclude excessive functional activity. Thyreoidectomy causes an increased output of thyreotropic hormone, and this has been put forward as an explanation of the severe exophthalmos which sometimes develops after that operation. As the exophthalmos usually present in thyreotoxicosis diminishes as a rule after thyreoidectomy, some other associated factor must be sought to explain the more typical cases of exophthalmos in toxic goitre. This raises the question as to whether there is an essential difference between severe progressive exophthalmos and the more usual form of proptosis seen in thyreotoxicosis. Clinically, there are well defined differences. Patients with severe progressive exophthalmos are older, and men are more frequently affected than women. Thyreotoxicosis is often not evident, and the patient may be myxœdematous. Pathological changes in the orbit can rarely be detected in the usual exophthalmos of thyreotoxicosis, but in the other condition they are usually found. However, one meets with patients who have both severe thyreotoxicosis and structural changes in the orbit. I believe that the origin of exophthalmos may be explained by the assumption that both thyreoid hormone and pituitary hormone are capable of causing exophthalmos. Thyreoid hormone does not cause structural changes in the orbit, but thyreotropic hormone will produce those lesions such as are found in the laboratory animal. In most cases of thyreotoxicosis both hormones are probably present in greater amounts than normal. If thyreoid hormone is having the predominant effect, the usual type of exophthalmos results. When pituitary thyreotropic hormone is the dominant agent, structural changes in the orbit are evident, and removal of the thyreoid, which causes a further increased production of pituitary hormone, causes further extrusion of the eyeball. This hypothesis, if correct, would also account for those cases in which some structural changes are present in the orbits of patients suffering from severe thyreotoxicosis, the degree of orbital change depending on the increase in thyreotropic hormone.

Pathological Changes in the Orbit.

Pathological changes in the extrinsic ocular muscles were observed in all Naffziger's cases and most of the others reported in the literature. The muscles are greatly enlarged and pale, and occupy from five to ten times their normal volume. The earliest change is a slight swelling of the individual muscle fibres associated with a loss of definition of the transverse striations. Interstitial œdema is present also, and collections of lymphocytes, plasma cells, occasional polymorphonuclear leucocytes and immature fibroblasts are seen between the muscle fibres (Friedenwald). Later, many of the muscle fibres are destroyed and replaced by fibrous tissue, in which collections of inflammatory cells are usually encountered. Some writers, including Brooks, have commented on the similarity of the muscle changes in this state to those occurring in Volkmann's ischæmic contracture, but in this latter condition gross macroscopic swelling of the muscles is less in evidence. The arterioles often exhibit periarteritis and endarteritis with surrounding lymphocytic and plasma cell infiltration. The histological changes suggest that either some toxic agent or an interference with the blood supply impairs the nutrition of the muscle fibres and is responsible for their œdema and ultimate destruction. The inflammatory cells may be due either to the liberation of the products from disintegrating muscle cells or to a response to the "toxic" agent. Examination of other skeletal muscles reveals no pathological change as a rule. Similar lesions in the extrinsic ocular muscles have been found in "idiopathic" exophthalmos, in *myasthenia gravis*, and in cases in which pronounced exophthalmos preceded the other clinical manifestations of thyreotoxicosis (Naffziger, Smelser, Marine); other workers have seen identical histological changes in animals injected with thyreotropic hormone, and in addition have described lesions in the lachrymal gland and orbital fat. The cause of the proptosis is the greatly increased bulk of the orbital contents, and the extrinsic ocular muscles are in a large measure responsible for this.

Clinical Features.

The ages of recorded patients vary between nineteen and sixty years. Brain, who studied 31 cases, found that in over 80% the patients were aged forty years or over. The age incidence therefore is greater than that of patients with the usual degree of exophthalmos found in thyreotoxicosis. Only 40% of the latter are over forty years of age. Also, the sex incidence is different, as a relatively larger number of males suffer from extreme degrees of exophthalmos. In Zimmermann's series of eight patients four were males, while in a series reported by Thomas and Woods there were eleven males and four females.

Recent experimental work by Marine strongly suggests that the high incidence in men is associated with the androgenic activity of the testes.

The onset is usually gradual over weeks or months. In post-operative cases the exophthalmos is usually pronounced between three and nine months after the operation, but may be observed as early as two weeks or as late as two years. Some patients show evidence of proptosis prior to the subtotal thyreoidectomy for an associated toxic goitre, and rapid protrusion of the eyes may follow. At first one eye may become prominent, then a little later the other protrudes or unilateral exophthalmos persists. Usually both eyes are involved to a considerable degree.

In Naffziger's first case the eyes protruded two centimetres beyond the average normal position. This is remarkable when one reflects on the observations that in the usual case of exophthalmos associated with thyreotoxicosis the protrusion is on an average only three millimetres more than normal.

As a rule the classical "signs" of both exophthalmos and lid retraction can be elicited. Rarely bilateral ptosis is present.

Conjunctivitis frequently accompanies the high degrees of exophthalmos and is associated with pronounced lachrymation. In such cases the œdematous conjunctivæ may be thrown into folds at the canthi, this being especially evident on side to side movement of the eyes. Chemosis and œdema of the lids follow as the condition progresses. Aching pain is often felt in the region of the orbits, and there may be tenderness where the superior oblique muscles are attached to the eyeball. The insertions of the recti muscles are sometimes visible, and the lachrymal glands are often palpable. Attempts to push the eyes backwards meet with considerable resistance, and discomfort or pain is produced if this is done.

Eye movements are generally impaired, and in some cases diplopia is a leading symptom. Naffziger found upward movement most affected, downward movement least, and side to side movement moderately impaired. The power of convergence is greatly limited. Weakness of the individual eye muscle is rarely present.

Corneal infection with ulceration is a grave complication, and if any associated thyreotoxicosis has been treated, it may be an indication for orbital decompression. No doubt the most important factor in causing corneal infection is the inability of the patient to approximate the lids, and so moisten and protect the cornea. Sometimes corneal anæsthesia may be an additional factor, for unappreciated trauma is not heeded.

Some patients with exophthalmos develop a degree of intolerance to light even before conjunctivitis causes severe photophobia. Albers and Sheard, who recently investigated this phenomenon in the usual cases of exophthalmos with toxic goitre, found that it was independent of the visual spectral change—that is, the quality of the light—and that it was improved after thyreoidectomy. Probably retraction of the eyelids gives rise to the condition by completely exposing the iris to light.

Changes in the retina and chorioid are not commonly seen, but papillœdema is more frequent.

Sattler found eight cases of retrobulbar neuritis in the literature and reported one instance himself. Foster Moore and others have noted its occurrence.

Optic atrophy has been seen and regarded as being due to the forward dislocation of the eyeball.

Papillœdema occurs apart from associated tumours or other pathological lesions and must be regarded as the important indication for orbital decompression. It was present in half Naffziger's cases and did not exceed four diopters. Optic atrophy and blindness follow the papillœdema if it is not relieved by operation.

Retinitis pigmentosa has been reported in one case in which the proptosis was pronounced.

The ultimate fate of a patient with severe progressive exophthalmos is uncertain, but frequently one or both eyes are lost as as result of corneal ulceration. Not uncommonly a septic meningitis occurs and terminates fatally. In many cases in which severe exophthalmos is present without evident thyreotoxicosis the condition remains stationary or recedes, even after a high degree of exophthalmos has been attained. In one case reported by Moore the condition remained unaltered for twenty-four years. On the other hand, in some instances the eyeballs are extruded with remarkable rapidity. In one case in which corneal ulceration necessitated the removal of both eyes, the remaining orbital contents continued to hypertrophy, and the fat and connective tissue finally projected between the lids. A biopsy was performed on this projected tissue, which proved to be histologically normal, the conjunctiva alone showing lymphocytic infiltration. Such a case illustrates the difficulties involved both in prognosis and in treatment.

Treatment.

Prior to 1930, many methods of treatment were tried and found either useless or inadequate. Among the more significant of these were cervical sympathectomy, deep X-ray treatment of the orbit, suture of the eyelids or other plastic operations upon them, and the oral administration of iodine or thyreoid extract.

As shown by Naffziger, orbital decompression is the only satisfactory method of treatment for severe progressive exophthalmos.

Iodine had been given to some patients in the mistaken belief that they were still suffering from thyreotoxicosis, but there was no improvement. Thyreoid extract has also been used in the treatment of the associated post-operative myxœdema; but while the eyes of some patients have not been noticeably affected, others appeared to be made worse by its administration.

Deep X-ray treatment has been given to the orbit, but without demonstrable improvement. Suture of the eyelids as a temporary measure for protection of the eyes may be advisable in some instances, but in the worst cases increasing proptosis forces them apart and the disease runs its course unchecked.

In 1930 Naffziger performed orbital decompression on a patient suffering from extreme exophthalmos, papillœdema, rapidly failing vision and greatly restricted eye movement. By means of a right frontal approach the greater portion of the orbital roof on that side was removed to give maximal decompression of the orbital contents. "The bone opening was carried mesially as far as the ethmoid and sphenoid cells, anteriorly as far as the frontal sinus, and posteriorly to the great wing of the sphenoid; laterally, the entire plate was removed." The orbital fascia was opened and the orbital contents were exposed. The retrobulbar space was found to be almost entirely filled with the greatly enlarged extraocular muscles. In view of the presence of papillœdema the optic foramen was also decompressed. This operation was followed by considerable immediate recession of the eye; the proptosis later increased a little, but never reached the degree seen in the preoperative stage. Vision and eye movements correspondingly improved. In 1938 this worker reported five subsequent cases, and the value of orbital decompression has been confirmed by the results of other operators.

Indications for Orbital Decompression.—When any associated thyreotoxicosis has been controlled the indications for orbital decompression are papillœdema, great impairment of eye movement, and progresive exophthalmos which threatens the loss of the eye from corneal infection.

Operative Technique.—In 1938 Naffziger, after a wider experience of orbital decompression, advocated a more extensive operation than formerly. Skiagrams are first taken to gauge the extent of the frontal and sphenoidal air sinuses and their relation to the orbital plates and optic foraminæ. If both eyes are involved a bilateral decompression is often performed in one stage. Not only is bone removed from the roof of the orbit, but the resection is carried laterally into the temporal fossa, and the lateral orbital wall is removed as far caudally as the maxillary antrum. When papillœdema is present the roof of the optic foramen and the superior orbital fissure is removed.

Results of Orbital Decompression.—Naffziger (1938) collected 30 cases, including eight of his own, in which the operation had been performed. The results were poor in only four instances and two deaths occurred. Unsatisfactory results are attributable in part to inadequate decompression, but the duration of the papillœdema and the extent of ocular muscle damage must be significant. In favourable cases the eyes return to the normal position or the exophthalmos is greatly reduced and eye movement is correspondingly improved. If the optic nerve fibres have not been destroyed normal vision is restored.

Summary.

1. An account is given of the ætiology, pathology and clinical features of both exophthalmos and lid retraction in goitre. As many of the classical signs of exophthalmos are in reality those of lid retraction, a plea is made for the more careful differentiation of the two conditions.

2. Many theories bearing on the causation of exophthalmos have been considered from the historical point of view and examined critically.

3. Evidence is presented which indicates that in goitre pituitary and thyreoid gland factors acting either alone or combined may bring about exophthalmos. In typical cases of toxic goitre the thyreoid hormone factor is dominant, and structural changes in the extrinsic ocular muscles are absent or minimal. The protrusion of the eyeball in such instances is due in a large measure to vascular hyperæmia in the orbit often associated with œdema of the periorbital fat. In man there is insufficient smooth muscle in the periorbital membrane or elsewhere in the orbit to produce exophthalmos. However, the excessive tone of the plain muscle in the upper eyelid (Müller's muscle of the upper eyelid) may result in lid retraction and apparent exophthalmos.

4. Paradoxical or severe progressive exophthalmos, which occurs both in toxic goitre and independently of it, is due to excessive secretion of pituitary thyreotropic hormone. The eyeball in such cases is as a rule extruded because of the increased volume of the extrinsic ocular muscles and lachrymal glands.

5. Methods of treatment of this rare but dangerous condition are outlined.

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EAR PICKING AND EYE CLEANING IN THE MIDDLE AND FAR EAST.

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Sydney.

Barbers of the Middle and Far East do not restrict their activities to the English routines of shaving and hairdressing. Indeed, many of the barbers in Singapore and Hong-Kong, China and Japan have no hesitation in audaciously advancing from what we regard as their recognized domains to closer preserves, the province of the legitimate aurist and oculist.

Eye Cleaning.

At Singapore I have seen and photographed (Figure I) a barber in the act of treating eyes. He used an instrument consisting of a stem of malleable metal inserted into a thin cane handle (Figure II). The free end of the stem was globular, the ball being slightly smaller than a match head. The stem was a twisted spiral of the same metal.

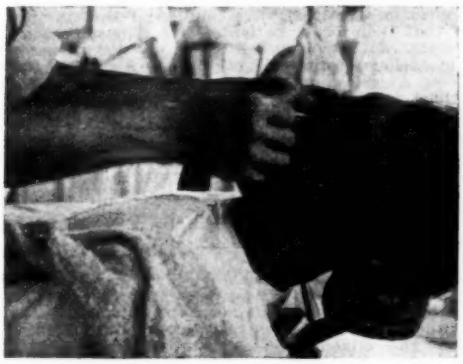

Figure I

The barber inserted the ball into the closed eye, at the inner angle, and, manipulating the lower lid, caused the ball point to run along the inferior fornix. At the same time, he exerted a light massaging and scraping action with the spiral part on the palpebral conjunctiva. Two or three times he repeated the manœuvre, dipping the instrument beforehand into a jar of water, presumably not fresh for each customer.

No medicaments were used, and the indication for the treatment, which did not appear to cause discomfort to the customer, was obscure. Probably it was a simple measure for the cleaning of the eyes, though it may be that a granular conjunctivitis had inspired the use of the spirally wound wire.

Figure II.

Ear Picking.

Ear picking is not the monopoly of the barbers, and vendors of picks encounter little difficulty in attracting buyers in the streets. When the barber undertakes the task he employs quite a little kit of tools for the operation. His instruments comprise a small razor, a pair of forceps, a probe curved on the flat, a curette and a twirler. The illustration (Figure III) shows the sizes and shapes of these instruments, made chiefly from cane and a light metal, such as silver, copper, aluminium, tin or white brass. Fine downy feathers fashioned into a pom-pom are the feature of the twirler, easily the least dangerous of the tools, but no less objectionable. Nevertheless, when the operator is experienced, relatively cautious and gentle, and when fortune watches over the patient, mutual satisfaction is achieved.

Firstly the barber shaves the tragal and antitragal hairs, then he extracts wax with the forceps. The curette and the probe are used according to the demands of the moment, and the rapid twirling of the pom-pom in the

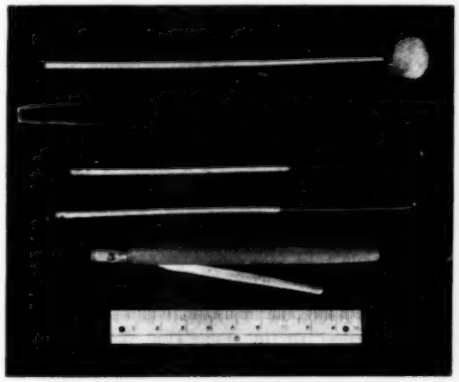

Figure III.

external canal completes the auricure. In spite of the hazards, the patient lives for the pleasures of the present, dismisses the fears for the future and lies in a semi-bewitched state, as may be gauged by his expression.

An unknown carver has recorded this expression in an uncommon carving in ivory (Figure IV). Buddha is shown

Figure IV.

receiving auricure, and the carver had captured the expression which frequently may be observed when the external auditory canal is stimulated gently. The

expression is difficult to classify; it compounds apprehension, concentration and reflection, appears to be reflex in character, and disappears as soon as the stimulation ceases. Possibly the grimace is a type of associated movement, examples of which are often manifest when an effort demanding concentration is made. For me the interest of the carving lies firstly in the carver's choice of a subject of medical interest, secondly in the wealth of expression which he has won from the ivory, and lastly in the implication that Buddha is not exempt from the visitation of minor annoyances; that Buddha, too, can suffer; that Buddha is real and not imaginary.

Hindus and Greeks.

Not many references can be traced in medical literature. Mukhopadhyaya,[1] in discussing the surgical instruments of the Hindus, mentions that the barbers of India now use an ear scoop for extracting wax. He classifies the instrument under the general heading of *Tala Yantra* or picklock-like instruments. The same writer also quotes from literature to indicate that the Greeks used similar instruments for the same purpose.

It can be seen that there are grounds for supposing a possible relationship between the barbers of India, China and Japan, and the working barbers contemporary with the barber surgeons of England in the fifteenth and sixteenth centuries. In the sense that these tradesmen did not limit themselves to shaving and hair-dressing, but strayed into neighbouring fields, East and West meet on common but thin ground.

Acknowledgement.

I wish to thank Mr. Woodward Smith, Department of Medical Artistry, University of Sydney, for the photographs of the carving and the instruments.

Reference.

N. Mukhopadhyaya: "The Surgical Instruments of the Hindus", Volume I, page 107.

Reviews.

A TEXT-BOOK OF MEDICINE.

As a straw showing which way the wind is blowing there is a heartening significance in the fact that October, 1940, was deemed an appropriate time to publish in Great Britain the fifth edition of the "Textbook of Medicine" (edited by J. J. Conybeare).[1] Every effort has clearly been made by the authors of the various sections to include all worth-while additions to medical knowledge available since the last edition appeared in 1939. They have done this without in any way disturbing the balance of what is unquestionably an excellent, highly condensed, yet readable and simple, medical manual.

"Conybeare's" has of recent years been so freely accepted by Australian students as a standard text-book, and it is finding its way to the bookshelves of so many general practitioners, that we feel justified in offering criticisms that we might not bother to raise in discussing a less important work. So when we read under the treatment of gastric and duodenal ulcers: "For six months after strict treatment has ceased it is wise to avoid meat completely but fish and chicken may be allowed", we wonder on what grounds the author perpetuates this red meat taboo. In Australia at any rate the matter is by no means as immaterial as it may at first sight appear, for if our future practitioners are going to preach this doctrine it must be well founded, since ulcers are common, fish is scarce, and chicken expensive.

Sulphapyridine has become so generally accepted in this country as the most effective drug for pneumonia that it is surprising to read in the chapter dealing with the treatment of this disease: "Sulphanilamide or sulphapyridine or a related compound should be given." Again, no one with any large experience of this form of chemotherapy would regard the suggested dosage as adequate to provide an effective blood concentration of the drugs—"1 grm. every four hours for forty-eight hours, followed by 1 grm. six-hourly for the next forty-eight hours". Since the average adult eliminates these drugs at a rate of approximately one gramme every four hours, even on this initial dosage the blood concentration would fall to practically nil six times in the twenty-four hours.

The intelligent use of digitalis is often of such vital importance that one might reasonably expect that when the dosage of this drug was under discussion some reference would be made to the fact that an international standard unit has been adopted that will digitalize a patient on a basis of one unit per 4·5 kilograms (ten pounds) of body weight and will be excreted at the rate of one unit each twenty-four hours. It is therefore somewhat disappointing to read: "In acute cases a single dose of 1-1½ dr. of the tincture may be given, followed six hours later by m. 20 t.d.s. until the full effect is obtained." Also it might go hardly with a student in his clinical *"viva"* if he accepted as correct that "m. 15 of a good tincture and 1 grain of powdered leaf may be taken as equivalent", since on an international basis the first is 1·0 digitalis unit and the second 0·6 unit. It is worth noting in passing that it is possibly due to the failure of Australian practitioners to obtain as good results with three grains of powdered leaf (1·8 units) as with forty-five minims of the tincture (three units) that their heart patients have almost ceased to have the benefit of the cheapest, least nauseating, and probably most efficacious of the digitalis preparations. In the United States of America, on the other hand, where the standard pill, tablet or capsule of the whole leaf is one and a half grains (one unit), this preparation is the most generally accepted method of digitalis therapy.

[1] "Textbook of Medicine", edited by J. J. Conybeare, M.C., D.M., F.R.C.P.; Fifth Edition; 1940. Edinburgh: E. and S. Livingstone. Demy 8vo, pp. 1151, with illustrations. Price: 24s. net.

CLINICAL DIAGNOSIS AND TREATMENT.

It is difficult to do justice to Dr. H. Joachim's "Practical Bedside Diagnosis and Treatment" in a short review.[1] It should be read by the readers of the review. In 821 pages the author treats of practically all conditions likely to be met by the clinician in private or in general hospital practice of many countries. As a physician he shows a nicely balanced judgement. Differential diagnosis and treatment in each case are considered with the correct proportion of sound surgical thought.

In the preface Dr. Joachim states: ". . . the ultimate aim of diagnosis is intelligent and judicious therapy . . . while I have a profound respect for the laboratory as a diagnostic aid, having been a laboratory teacher and worker, I have the feeling that routine laboratory surveys and fine combings must not usurp, displace or blunt the diagnostic skill of the senses and reasoning based on sound anatomical and physiological principles. As a teacher of medicine I have always stressed the clinical phases of disease. The laboratory must cooperate, collaborate and supplement, but should not dominate the diagnostic processes . . ."

The book is divided into 12 sections, each "system" being treated in detail. Some headings, at random, are: "Diseases of Infectious Origin", "The Heart, Pericardium and Blood Vessels", "The Alimentary Tract and Peritoneum". The author is up to date in his discussions on the ætiology of diseases and conditions, and also in treatment. Praise must be given the author for the thorough "Differential Diagnosis" in each case; his references to minor causative factors show that he has both common sense and a keen power of observation. The practical healer overcomes the scientist in his dictum (unfortunately not always followed): ". . . in pneumonia . . . frequent physical examinations are disturbing and harmful to the patient. Under no circumstances should elderly patients be permitted to sit up in bed during physical examination . . ."

Of the twelve good sections the following stand out: "Heart", "Metabolic and Deficiency Diseases" and "Central Nervous System". Dr. Joachim can combine terseness in enumeration of condensed facts with entertainment; therefore this book can be read with interest by student and graduate or used as a modern reference book. The publishers have produced a book well set up and well printed, with only one word ("edema") in the "American-English" manner of spelling.

[1] "Practical Bedside Diagnosis and Treatment", by H. Joachim, M.D., F.A.C.P.; 1940. London: Baillière, Tindall and Cox. Royal 8vo, pp. 844. **Price: 41s. net.**

The Medical Journal of Australia

SATURDAY, APRIL 5, 1941.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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THE WHITE MAN IN THE TROPICS.

The problem of tropic colonization assumes every year a greater and more urgent importance to Australia. What many writers fail to realize is that conditions in Australian tropics are widely different from those in territories climatically similar and that in consequence the problems must be solved locally and with little help from experiences in other lands. In all other tropic countries there is a large native population acting as a permanent reservoir of endemic disease and, further, the white man is a manager or overseer and the white woman is supplied with abundant and cheap coloured labour in her home. In Australia north of Capricorn the native population is sparse, endemic diseases have little menace and the whole work of the community is carried out by the white race, manual as well as administrative, unskilled as well as technical.

A vast amount of research work has been undertaken in an attempt to disentangle the effects of tropical diseases from the effects of climate alone. We have also only recently learned that malnutrition plays a much bigger part than was realized, for the absence of milk and many protective vegetables cannot fail, unless corrected, to lower vitality. The pity of it is that so many authors dealing with life in the tropics cannot perceive that medical and physiological considerations furnish the main key to the entire problem. The recently published book of A. Grenfell Price, "White Settlers in the Tropics", was reviewed favourably in this journal. The author, an Australian, has not been medically trained, but has made a praiseworthy effort to put the medical position before his readers; nevertheless the emphasis he places on disease and malnutrition is not as great as medical investigators would like. In his recently published book "Australia" Professor Griffith Taylor gives amongst a most valuable presentation of Australian geography, geology, climate, soil, primary and secondary industries, some pronouncements on tropical reactions which display a curious blindness towards the medical and nutritional facets of the question.[1] No doubt much of what Professor Griffith Taylor has been saying, and saying somewhat loudly for years, is perfectly true, namely, that comfort and economic possibilities will continue to keep Australians out of the tropics so long as there is room for expansion of population in the extratropics; but he advances arguments, surely threadbare by this time, which indicate a veritable mental amaurosis on medical factors. It is quite beside the purpose to give statistics concerning the condition of people living on the coast of the Gulf of Mexico as applicable to those portions of Australia with similar climate. Professor Taylor argues that the Australian tropics are as bad climatologically as tropics anywhere, and he may be perfectly correct in this contention; but it would be interesting if he would explain why sunstroke or heatstroke is so rare in Australia and so common in other tropic lands. Indeed the prevalence of fatal sunstroke in tropical Asia, Africa and America and the absence of anything like this phenomenon in Australia have suggested to many Australian medical thinkers that the so-called sunstroke of other lands is a fulminating infection and is not caused by the physical conditions of temperature, humidity and ventilation with or without insolation. Both Dr. Price and Professor Griffith Taylor refer to the constant attention to health matters and the perpetual vigilance against malign agencies which are necessary in tropic residence; but in Australia no such worries afflict the inhabitant. The Queenslander will saunter out in the sun with any makeshift of a hat or none at all, and if he is bitten by a mosquito will not rush to his bottle of quinine tablets—he usually has none. His life, with its abundant open air and with body unencumbered by heavy clothing, is really nothing like so artificial as the life of a resident in a colder country. Professor Griffith Taylor confesses that in his Toronto home he is compelled to resort to central heating for eight months of the year. Nothing will ever convince the Queenslander that life in a stagnant thermostat is not far more artificial and far more depressing than life amid the sunshine and free undoctored air of his homeland. One has only to contrast the bronzed manhood of Queensland with the pallid youth of northern America, just emerging in early summer into the pure air, to realize that the criticism should be reversed. The civilizations of Egypt, Phœnicia, Greece, Carthage and Rome arose in a Mediterranean environment with which temperate Australia has many affinities. The Roman urge for conquest did not extend north of the Rhine, the Black Sea and the Tweed for the sufficient reason that such northern countries, with their desperate winter, were in contemporary opinion fit only for barbarians. Where the olive, vine and fig did not grow could not be a home for civilized man. We have shifted our frame of reference since that time and the future may have another transvaluation in store.

The most recent contribution to this vexed problem of man in the tropics comes from the medical school of the University of Queensland and is very welcome.[2] Professor Douglas Lee, in his brochure "A Basis for the Study of Man's Reaction to Tropical Climates" sets out in perfect scientific temper and with proper technical treatment the

[1] Griffith Taylor: "Australia: A Study of Warm Environments and their Effect on British Settlement", 1940.

[2] Douglas H. K. Lee: "A Basis for the Study of Man's Reaction to Tropical Climates", University of Queensland Publications, December 30, 1940.

known facts about the functioning of the human body in a tropical environment. Anyone unversed in medical science seeking political ammunition in this scholarly work will be disappointed, for the absence of imaginative handling and of plausible suggestions and inferences, though it makes reading difficult, is a source of real strength. We trust that Professor Lee will be allowed to devote himself wholeheartedly to this wide and varied field of investigation and that he will remain untrammelled by political interference. The greatest enemy of science today is the politician with power to reward, degrade or dismiss. An optimism that is dictated or bought must in the long run be as detrimental to Australian interests as a pessimism founded on ignorance.

Current Comment.

ENDEMIC FLUOROSIS.

THE remarkable disorder known as fluorosis was commented on in these pages on January 15, 1938. It was then pointed out that the generally recognized sign of fluorine intoxication was mottling or banding of the dental enamel in children, but that in certain districts, notably Nellore, South India, where the ingestion of fluorides in the drinking water continued over many years, the skeletal system of adults was seriously damaged. The bones become dense, and osteophytic outgrowths appear at points of stress, such as the sites of muscular attachments. Similar manifestations of the disease have been observed in other parts of the world, notably in Denmark, among workers in cryolite (an ore consisting mainly of sodium-aluminium fluoride). Recent investigations have shown that fluorosis occurs over a much wider area in South India than the comparatively small district of Nellore. T. N. S. Raghavachari and K. Venkataramanan have recently carried out a survey of an extensive area in South India with a view to ascertaining the fluoride content of the drinking water and devising some method of treating water so as to remove the contamination.[1] Specimens of rock obtained from some of the severely affected areas were found to contain "fairly large" amounts of fluorite and fluorapatite. No correlation was found between the fluoride content of water and the other main features, such as chloride and silica content, hydrogen-ion concentration *et cetera*. Generally they found a relationship between the concentration of fluorides in the water and the severity of the toxic manifestations. They were unable to find any chemical process suitable for the purification of water on a large scale in the rural areas that they investigated. They note, however, that a proprietary substance known as "Defluorite", which consists largely of alumina, is effective; but large amounts of it are required, and its use may be too costly.

C. G. Pandit, T. N. S. Raghavachari, D. Subba Rao and V. Krishnamurti have made a study of the possible factors concerned in the development of the dental and bony lesions characteristic of fluorosis.[2] They noted several features of interest. Even if the fluoride content of the water was high, the incidence of the disease was lower and the severity less where the diet was more nutritious. In communities where the food was poor, the incidence was higher and the severity greater, even though the fluoride content of the water was comparatively low. The most important dietary factor seemed to be vitamin *C*. Where two villages had water supplies of about the same content of fluorides, the one that was better provided with vitamin *C* was less affected with fluorosis, as to both incidence and severity. Pandit and his colleagues found that an adult must reside in an endemic area for at least fifteen years before he showed signs of the disease in its chronic form. The severity and site of the lesions depend to some extent on occupation. For example: the incidence of advanced fluorosis was 9% among clerks, teachers, merchants and artisans, as against 16·2% in labourers. The limb most subject to muscular strain is the first affected; for example, in basket weavers the left wrist and arm are the first to show signs of the disease. In the late stages the spine becomes rigid and the thorax fixed and the joints of the extremities ankylosed. In addition to the drinking water, Pandit and his colleagues mention the possible influence of the common grinding stone, made from local rock, containing fluorides, and used for grinding meal.

Experiments in the production of fluorosis in the laboratory animal have been made by C. G. Pandit and D. Narayana Rao.[1] Two monkeys (*Macaca radiata*) were given a normal diet containing vitamin *C* and no fluorine; two were given a similar diet and a measured amount of sodium fluoride by stomach tube; two others were given sodium fluoride and a diet deficient in vitamin *C*. The first two monkeys continued in good health; the second two absorbed less fluorine and excreted less in the urine than the third two. Bony changes of fluorine intoxication were revealed by radiological examination in all the animals receiving sodium fluoride; but they were much more pronounced in those whose diet was deficient in vitamin *C*. The paper by Pandit and Rao is illustrated with reproductions of skiagraphs, taken five months after the commencement of the experiment, showing these remarkable changes.

Two of the monkeys died during the experiment. No abnormality of their organs was noted, save slight hyperplasia of the thyreoid in one and cloudy swelling of the kidneys in the other. The concentration of fluorides in the organs had not been determined at the time the paper was submitted for publication.

Fluorosis is not merely a medical oddity. It has been observed in various places. Its incidence throughout the world may be very much higher than is suspected at present. It scarcely seems possible that the disease in its advanced form could remain hidden for long in a highly civilized community; but it may well be that minor manifestations are often seen without being recognized.

[1] *The Indian Journal of Medical Research*, October, 1940.
[2] *Ibidem.*

HYPERTENSION AND RENAL ISCHÆMIA.

IN 1934 H. Goldblatt demonstrated that partial constriction of the renal arteries of dogs resulted in a rise of blood pressure. Four years later Taquini showed that by completely occluding the renal blood flow for a few hours and then allowing it to become reestablished, a similar rise of blood pressure occurred. It had long been recognized that hypertension in man was often associated with severe renal damage, and these two workers demonstrated graphically that damage to the kidney can be the causative factor in the production of this elevated pressure. It has been believed for some time that the pressor substance produced in the ischæmic kidney is renin, a belief that is borne out by recent experimental work by M. Prinzmetal, H. A. Lewis and S. D. Leo.[2] They investigated acute complete renal ischæmia. The pedicle of one kidney of an animal was completely occluded for several hours, and the organ was then perfused with saline solution. The perfusate, when injected into other animals, caused a considerable rise in blood pressure and contained a substance which in all properties investigated corresponded exactly with renin. This was not contained in significant amounts in the perfusates of the normal kidneys of the same animals. Similar complete ischæmia of a limb or of a gravid uterus failed to produce a similar pressor substance. It seems that as a result of renal ischæmia, acute and complete, and probably also chronic and partial, more renin is formed in the kidney or that which is present becomes more readily available for extraction.

Hypertension is so common a problem and its cause has so long been elusive that Goldblatt's work aroused tremendous interest, and very soon received corroboration

[1] *The Indian Journal of Medical Research*, October, 1940.
[2] *The Journal of Experimental Medicine*, December 1, 1940.

by the reports of cases in which hypertension associated with disease of one kidney was relieved by surgical removal of that organ. When a number of such cases had been reported it was plain that the next step for clinicians to take was a critical review of the incidence of hypertension in those renal diseases amenable to surgery, noting particularly the effect on this hypertension of the removal of a single diseased kidney. Such a review has recently been made by the Section of Urology of the American Medical Association. Papers were submitted by W. F. Braasch, W. Walters and H. J. Hammer, of the Mayo Clinic, and by E. G. Crabtree and N. Chaset, of Boston.[1] The first of these describes the incidence of hypertension in a group of 1,684 patients submitted to renal surgical procedures in the clinic, and the effect of operation on the blood pressure. A preliminary survey of patients suffering from any disease at all showed that among the 975 examined 20% had a blood pressure of more than 145 millimetres of mercury, a fact which must, of course, be taken into consideration when hypertension among renal patients is discussed. The incidence of this hypertension among non-renal patients fell chiefly among the higher age groups. On the whole, the incidence of hypertension among the 1,684 patients submitted to renal surgical operations was no higher than among this control group. Atrophic pyelonephritis was the disease most often associated with a raised blood pressure, 46·5% of the patients having a pressure in excess of 145 millimetres of mercury. The result of the removal of a single diseased kidney in this group varied, but a majority of those who had hypertension before operation were found to have a low pressure at follow-up examinations. Pyelonephritis without atrophy and sclerosis, acute cortical renal infection and perinephric abscess were not found to be causes of hypertension. Renal calculus seemed to cause a rise in blood pressure only if accompanied by extensive infection and sclerosis of the kidney. The incidence of hypertension was not greater among patients suffering from renal tuberculosis or hydronephrosis than it was among the control group. A slightly higher incidence in the group with carcinoma of the kidney was probably due to the higher average age of these patients. A follow-up study was made on 198 patients in whom hypertension was present and on whom operation was performed. The blood pressure became normal after operation in 65 of these and remained normal for a year or more. The authors predict that hypertension will be relieved by operation in approximately 70% of cases in which it accompanies atrophic pyelonephritis, in 50% of cases in which it is associated with renal tuberculosis, and in about 25% of cases in which it accompanies renal stone, hydronephrosis or tumour. In some patients it was found that the hypertension returned after being absent for as long a period as a year. Crabtree and Chaset studied the incidence of hypertension among 150 patients with severe unilateral renal lesions, and performed careful histological examinations of the kidneys removed at operation to determine the degree of arterial obliteration and consequent renal ischæmia present. Hypertension was not a common finding; only 14 patients were hypertensive, and the average blood pressure was not above the normal for any age group. Vascular changes, which have been considered causative factors in the production of hypertension, were, however, found in 58 of the 150 patients. These changes were of a degree sufficient to cause some renal ischæmia. This number is much higher than the 14 in whom hypertension was actually found. The discrepancy becomes still more striking, for three of the fourteen hypertensive patients did not show histological evidence of renal ischæmia. Nephrectomy was not followed by striking changes in blood pressure. A definite fall occurred in one patient. Four others had slight but insignificant falls, and some actually had a rise in pressure following the operation. The authors' conclusion is that hypertension is not an indication for operation, which should be reserved for those cases in which a definite surgical indication for operation exists. They think that their investigation has shown that the anatomical and pathological changes in the kidney are not the important factors in determining the onset of hypertension, and suggest that it is due to some physiological element as yet not understood.

These results are not really surprising. If hypertension had been a consistent finding in those renal diseases which can be treated by nephrectomy, it is unlikly that it would have taken the work of Goldblatt to arouse the interest that is now being shown. Indeed the survey from the Mayo Clinic shows a rather higher incidence of hypertension than we would have expected, even when we allow that in some of the cases it was not caused by the anatomical changes in the kidney that indicated operation. These two surveys show one thing; it is not the anatomical changes, either macroscopic or microscopic, known to occur in surgical diseases of the kidney that are the cause of hypertension. The question is by no means closed yet, and there is room for many more surveys of the type reviewed here. The cause of hypertension may well be found to originate in the kidney, but the problem is still physiological.

[1] *The Journal of the American Medical Association*, November 30, 1940.

REGENERATION IN CARDIAC MUSCLE.

THE doctrine that the cells of the "specialized" tissues of the body are incapable of self-regeneration and proliferation in the manner of the cells of "less differentiated" tissues is now long out of date, as E. S. J. King, of Melbourne, points out in a recent communication on regeneration in cardiac muscle.[1] For some time such capacity was denied the more specialized epithelia, such as the cells of the liver and kidney; but when hepatic and renal proliferation was recognized, the original doctrine was still applied to other tissues, notably muscle and nervous tissue. Then hyperplasia of smooth muscle and, later, regeneration of voluntary muscle were described; but hyperplasia of cardiac muscle has never been unequivocally demonstrated.

King has made a histological study of a wound of the heart in a young adult; the wound was sutured surgically, but death occurred several days later from streptococcal septicæmia. King claimed that he observed proliferation of the cardiac muscle in the vicinity of the wound, because he saw and photographed what he believed to be splitting of the muscle fibres, as indicated by the size and arrangement of the fibres and their relationship to the capillary vessels, protoplasmic outgrowths from the ends of damaged fibres, the presence of double nuclei in some fibres and the presence of mitotic figures. He did not, however, suggest that such proliferation played any considerable part in the healing of an injury to the heart, which occurs by the usual connective tissue proliferation.

However, the opinion that hyperplasia of cardiac muscle does not take place will die hard. An editorial note appended to King's article challenges his conclusions. The splitting of the muscular fibres and their apparent arrangement in pairs might be an artefact, the editors write; the protoplasmic outgrowths and many of the changes seen in the nuclei might be the result of the infection and of degeneration; the double nuclei might be Purkinje fibres or might be due to a superimposition of nuclei. They add that the author had considered these explanations and rejected them; but still to them the evidence seemed inconclusive. The editors of medical journals often feel a certain scepticism concerning some of the conclusions of their contributors; it is refreshing to see this scepticism authoritatively and constructively expressed, even if in this particular instance some of the criticism seems a little captious. It may well be that King will not have another opportunity to examine the question; on future occasions, we hope, he will have the greater satisfaction of saving the lives of his patients wounded in the heart; but the brief controversy which we have quoted will surely awaken wide interest and it should not be very long before the question is determined.

[1] *British Heart Journal*, July, 1940.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Corneal Ulcers and "Prontosil".

ROGGENKAMPER (*Klinische Monatsblätter für Augenheilkunde*, August, 1939) has confirmed Thiel's and Heinz's favourable opinions regarding the value of "Prontosil" treatment in corneal ulcer. He administered the drug in tablet form and intramuscularly as "Prontosil soluble". He considers that as the drug is unusually pharmacologically indifferent it can be used in eye drops or in subconjunctival injections. He has often used the latter method and believes that the former should be tried. Particularly with *ulcus serpens* has he obtained good results. In almost all cases a hypopyon was present, and without exception quick healing occurred and little scarring was left. The whole of the cornea was involved in one eye and even a cataract ensued, yet after its removal the cornea was so clear that vision of $^{6}/_{18}$ was obtained. In several instances he thought that perforation was inevitable; but this was obviated by the rapid healing. Other types of ulceration were benefited, but not to the same extent. In two cases of obstinate parenchymatous infiltration associated with ulceration the condition cleared up with each injection, even though it had persisted for several weeks previously. The author thinks that "Prontosil" may make operative measures unnecessary in the treatment of exogenous infections of the cornea.

The Eye of Argyropelecus Hemigymnus.

FILIPPO CONTINO (*Archiv für Ophthalmologie*, April, 1939) describes in great detail the morphology, structure, development and refraction of the eye of the silveraxe fish, a teleostean, found at a depth of 400 to 2,900 metres in the Mediterranean Sea and the Indian Ocean. Like other deep-sea fish, it possesses telescopic eyes and a light organ. Its body length is three to four centimetres. In the larval stage the eye is not telescopic, in marked contrast with *Scopelus caninianus*, which is furnished with a telescopic eye in the larval but not in the adult stage. The author was able to obtain abundant live material in the adult stage as well as embryos up to seven days hatched in the laboratory and older larvæ caught off Messina. Fresh unfixed globes were examined *in situ*. Others were fixed in 3% formalin in sea water and examined macroscopically with and without bleaching with free chlorine. Serial sections were made of fixed material stained in bulk. The telescopic eyes look directly upwards and are each provided with six muscles. They rotate through an angle of 45° in an antero-posterior direction only. In the fresh state the spherical lens almost completely fills the anterior chamber. When fixed, owing to shrinkage of the vitreous, the lens falls backwards. The *retractor lentis* muscle is easily demonstrated macroscopically. There is no hyaloid system and the retina contains only rods. The sclera is cartilaginous and the cornea homogenous in structure and of uniform thickness, except in the centre, where it is a little thinner. The gradual development of the telescopic eye is traced from the embryonic to the adult stage. The optical constants are carefully worked out, as the eye is much too small for retinoscopy. *Argyropelecus hemigymnus* has a myopia of 64 diopters with a far point of 20·8 millimetres. This is removed to 42 millimetres by the action of the *retractor lentis* in pulling the lens 0·25 millimetre backwards. The visual field in each eye covers an angle of 90° antero-posteriorly and 50° laterally. As the eyes are actually touching in the mid-line, the fish possesses a small binocular field. Because of its poor vision the eye can be of little use in the search for food. It may be of some sexual significance and of use in seeking out the ventrally situated light organ of its mate. Hence the term "telescopic eye" is truly ironic. As only the short waves of the spectrum, and particularly the ultra-violet waves, penetrate to the depths of the ocean, it may be that the eye responds to them. These wave-lengths, however, are absorbed by the lens and do not reach the retina. Nevertheless, it is possible that deep-sea fish use not the ultra-violet light, but the feeble fluorescence present in deep-sea water.

Nævus Flammeus of the Lids and Congenital Glaucoma.

PASCHEFF (*Klinische Monatsblätter für Augenheilkunde*, May, 1940) has described several examples of what have been called phacomatoses. The patients include a twenty-eight year old patient with a "port-wine stain" of the right lids, cheek and palpebral conjunctiva and hydrophthalmia of the eye of the same side. The cup was white and deep. Among the number is also a seventeen year old patient with a slowly increasing plexiform neurofibroma of the right upper lid and the right cheek. The lids could not be separated voluntarily. Radiography of the orbits and *sella turcica* failed to reveal any defects. The right eye was very enlarged and prominent, the pupil widely dilated and fixed. The disk was deeply cupped and the tension very high. The vision was reduced to $^{1}/_{20}$. These congenital tumours or phacomata may affect the lids, the iris, the retina and the optic nerve. In addition to hydrophthalmia the first variety may be associated with cataract, astigmatism and amblyopia. The other varieties are included in the syndromes described by von Recklinghausen, Bourneville and Sturge and Weber.

Relapses of Tuberculous Ocular Disease.

W. HALLERMANN (*Klinische Monatsblätter für Augenheilkunde*, April, 1940) does not agree that each relapse of tuberculous ocular disease means a fresh hæmatogenous metastatic infection. In only a very small number of patients with active gland processes has he observed relapses which aroused suspicion of fresh hæmatogenous dispersion three to four weeks after a period of debility and anorexia. The majority of the very frequent relapses must be classed as local relapses. Wegner has demonstrated by animal experiments that virulent bacilli are present in the eye even months after an apparent cure of an intraocular tuberculous focus. This explains the frequency of relapses and the intractibility of the disease. The author considers that the exudative type of irido-cyclitis and scleritis responds best in periods of weather with constant high pressure conditions, and that the proliferative and fibrous cases require stronger climatic changes. In the high-lying resorts the winter months appear to be especially favourable for effecting cures, owing to the clear dry air and the richness of ultra-violet rays, which are reinforced by snow reflection. These months are dangerous, however, in the low districts.

Clinical and Serological Diagnosis of Gonococcal Iritis.

E. J. SIEGERT (*Archiv für Ophthalmologie*, April, 1939) states that gonococcal iritis resembles the so-called rheumatic type in its sudden violent onset with pain and visual loss. These changes are much less when iritis is due to tuberculous or syphilitic infection. A moderate gelatino-fibrinous exudate of flaky structure and a pronounced tendency to synechia formation are present, and there is an absence of tubercle formation. In the Hamburg clinic a monarthritis was found in three of eleven cases, and rheumatic complaints were rare. The results of complement-fixation and flocculation tests were of confirmatory value. The author reports his experience with specific treatment, including "Gonoyatren" and "Arthrigon".

Ocular Tuberculosis.

W. WEGNER (*Klinische Monatsblätter für Augenheilkunde*, April, 1940) considers that the reports in the literature of cures of ocular tuberculosis are too optimistic because of inadequate observation, that this disease is one of the most common causes of blindness, and that no advance in treatment will be made unless the aftercare of patients is systematically undertaken. He arranged with the biggest social insurance firm in Germany, the Imperial Insurance Society for Employees, to classify all patients with ocular tuberculosis who had been seen in the previous six or seven years. These patients would be reexamined at the end of each six months and, if necessary, treatment arranged. This work was considered to be particularly valuable, as more than a third of all such patients came from strongly tuberculous families.

OTO-RHINO-LARYNGOLOGY.

Complications of Surgical Treatment of Acute Mastoiditis.

G. F. HARKNESS (*Archives of Otolaryngology*, November, 1940) discusses complications of surgical treatment of acute mastoiditis and concludes that chemotherapy has changed the preoperative and post-operative pictures of the condition. Its potentialities for good outweigh its potentialities for harm. The latter are real, and contraindications to chemotherapy must be recognized. Possible masking is to be considered. Indications for surgical treatment of the mastoid cavity, the lateral sinus and the petrous pyramid remain; but chemotherapy may prevent development of the pathological condition or cause regression so that operative intervention is unnecessary. Sulphanilamide and sulphapyridine, the action of which is probably limited to the bacteriostatic effect, accomplish more the earlier they are used. They are not a cure-all. Criticism based on the

possible masking effect is unjust when therapy is not continued after the clinical symptoms subside. Therapy, barring contraindications, should continue for ten days after apparent clinical and bacteriological recovery.

Tumours of the External Auditory Canal.

HORACE E. MITCHELL (*Archives of Otolaryngology*, November, 1940) states that tumours of the external auditory canal are so rarely encountered in clinical practice that they are bound to arouse unusual interest. The age of patients with malignant tumours of the external auditory canal corresponds to that of patients with malignant growths elsewhere in the body. Instances have been recorded of occurrence in patients from youth to extreme old age; but the majority of patients are in the fifth or sixth decade of life. The cases of malignant disease of the external auditory canal recorded in the literature seem to be evenly divided between men and women. The most important types of malignant disease of the external auditory canal are: (*a*) carcinoma, (*b*) sarcoma, (*c*) rodent ulcer and (*d*) endothelioma. The most common malignant growth is squamous carcinoma, although a number of cases of adenocarcinoma have been observed. Several cases of prickle-cell carcinoma have also been reported. Scott and Colledge suggested that primary carcinoma should be divided into two main types, identified by early or late involvement of the tympanum. Some growths are recorded as arising in the middle ear: but Scott and Colledge cited Ernest West's statement that all squamous carcinoma arises in the fundus of the external meatus and spreads inwards, involving the tympanum. The meatus appears to be the starting point in the majority of cases. In the second type, that in which the tympanum is not involved until late in the course of the disease, primary ulceration in the meatus usually attracts the patient's attention by causing pain or irritation before the growth has spread widely. Sarcoma and endothelioma present few clinical features to distinguish them from carcinoma, and the differentiation can be made only by histological examination. Other symptoms are ulceration, aural discharge, deafness, giddiness and in late stages facial paralysis. Other factors which should increase the suspicion of malignant growth are: (i) toughness of the granulations when touched with a probe, (ii) recurrence after removal and tendency to bleed easily, (iii) necessity for persistent treatment, (iv) pain on chewing, and (v) granulations or papillomata attached to the deep meatal wall. The diagnosis of malignant disease can be made only by histological examination. All cases in which granulations or a persistent ulcer are present, especially in patients more than forty years of age, require consideration of malignant disease. If early diagnoses are to be made, the possibility of malignant growth should be considered also in all cases of long standing chronic suppuration of the ears, because the records show that a great proportion of patients with malignant disease have had aural discharge for years. The treatment of malignant tumour of the external auditory canal is surgical removal, usually accompanied by radical mastoidectomy. Some authors advocate the radical operation in all cases, and others believe that when there is no involvement of the tympanum a radical procedure is not necessary and that the growth can be removed through the meatus. Some form of irradiation, either with radium or with X rays, is usually employed after surgical intervention. The most common benign tumour of the external auditory canal is osteoma. According to Banyai and Janota, osteoma of the auditory canal appears usually after puberty and is almost always unilateral. The incidence is much greater in males than in females. The reason for this predominance is unknown. The incidence also is greater in northern lands, which suggests that there may be a metabolic factor in the causation. Other benign growths which have been reported in the literature include xanthoma, dermoid teeth, von Recklinghausen's disease, pigmented and non-pigmented nævi and organized blood clot. That chronic infection has some relation to the development of neoplastic tumours is suggested also by the fact that a predominant number of patients with malignant disease of the external auditory canal have had aural discharge for years. Many malignant lesions have appeared clinically as simple polypi, and only histological examination has revealed their true nature. If patients themselves were educated to the potential seriousness of continuous discharge from the ear, and if otologists were more alert to suspect malignant disease, it might be that a greater proportion of cases would be discovered and treated earlier. The rate of growth of auditory tumours is slow and the lymphatic spread often late.

Suppuration of the Petrous Pyramid.

ROBERT L. MOORHEAD (*Archives of Otolaryngology*, November, 1940) discusses suppuration of the petrous pyramid and agrees that in the treatment of this condition no single procedure is suitable for all cases. When pyramid signs are present at the time of invasion of the middle ear, a simple mastoidectomy should be performed and a careful search for tracts made during this procedure. The result of this operation should be awaited before any further surgical intervention is attempted. Undoubtedly many patients recover spontaneously after a simple mastoidectomy. Two years ago the author advocated an approach to the pyramid along the floor of the middle fossa and the avoidance of the radical mastoidectomy. By this approach it is possible to reach the pyramid, drain the infected focus and have the patient recover with an intact middle ear and the preservation of good hearing. Objection has been made to this route on account of the likelihood, first, that the dura will be torn while the elevation is being carried out or while the focus along the dura is being drained, and secondly, that the petrosal nerve will be torn. These objections are theoretical rather than practical, for in no case have bad results been observed which could in any way be ascribed to this method of approach.

ORTHOPÆDIC SURGERY.

Fractures in the Neck of the Femur.

EDWIN O. GECKELER AND ALFRED TUTTLE (*Surgery, Gynecology and Obstetrics*, January, 1941) describe a method of fixing fractures of the femoral neck by two screws inserted subcutaneously under observation with the fluoroscope. Originally the main difficulty of the operation, since lateral observation of the femoral neck was impossible by this means, was to obtain accurate lateral placement of the screws. Errors were made owing to two factors: (*a*) the difference in the degree of internal rotation to which every leg can be turned, and (*b*) the considerable anatomical variation in the angle at which the neck leaves the shaft, as compared with the plane of the patella. To overcome the difficulty, a block supporting a steel guide pin and a cannula parallel with the pin is now used. After reduction by the Leadbetter method the limb is held in wide abduction and internal rotation. Protection against the X rays is provided by a suitable filter and apron. By intermittent use of the fluoroscope the line of the femoral neck is determined and a small transverse incision is made through the skin and deep fascia of the thigh. The guide pin is now directed along the anterior surface of the neck until its point impinges on the head of the femur. Inward pressure on the cannula will now bring this immediately along the line of the neck. A drill is inserted through the cannula and a hole is bored across the outer fragment. A steel screw, of suitable length, is then driven across the fracture line. A second screw is inserted above the first location by partially withdrawing the guide pin and replacing it half an inch to three-quarters of an inch distant from, and parallel with, the first. Each step is checked through the fluoroscope. After insertion, antero-posterior and lateral skiagrams are taken. If the position is satisfactory, the instrument is withdrawn and the skin wound is closed with Michel clips. If not, the necessary withdrawals and adjustments can be made. The patients are allowed to sit over the side of the bed on the first day after operation, and are gradually encouraged to stand without aid, the foot of the affected leg merely touching the floor. Weight-bearing without crutches is not allowed until X-ray examination reveals union. Fifty patients were treated, and the average age was seventy-three years. The average stay in hospital was twenty-four days, and the average time for operation was twenty-three minutes. There were three operative failures and no operative deaths.

Spontaneous Fracture of the Fibula.

H. JACKSON BURROWS (*The British Journal of Surgery*, July, 1940) records two cases of apparently spontaneous fracture of the lower third of the fibula. One patient was an elderly woman of sixty-one years, whose occupation involved much walking. The other was an active public school boy of seventeen. Both fractures were subperiosteal in type, with no displacement. In neither case was there any history of injury. Radiographs showed poorly calcified callus. Before treatment, pain, swelling and tenderness were the symptoms. Adhesive elastic bandaging was applied for six weeks in the case of the woman, and in the case of the youth restriction of activity was recommended. Good recovery resulted in both instances. The author suggests that the fractures are similar in type to the march fracture of the metatarsals. He refers to Hopfengartner's series of fractures of the upper third of the fibula in infantrymen without history of injury, and which were termed by Köhler "recruit's disease".

British Medical Association News.

VICTORIAN BRANCH NEWS.

THE following statement is published at the request of the Council of the Victorian Branch of the British Medical Association for the information of members.

The Motor Car (Third-Party Insurance) Act 1939.

The *Motor Car (Third-Party Insurance) Act* 1939 of Victoria, recently proclaimed, provides that on registration during 1941 and subsequent years, every owner shall effect an insurance policy in accord with the various provisions of the Act by paying the appropriate premium to the Chief Commissioner or by effecting a "proper" policy with an "authorized insurer". This premium will indemnify the owner against liability for third-party injury, but only if the liability is one which would have arisen in the absence of any contract of insurance, that is, "negligence" must still be proved by the person injured.

Liability is limited to £2,000 in respect of any one individual injured and £20,000 in all for any one accident.

The Act also provides for claims to be made against the "authorized insurer" should the owner die and also for claims against a nominal defendant should the car not be identified.

Any owner who permits the use of an uninsured car is liable to a fine of £100 or three months' imprisonment.

The Act makes provision for the payment to public hospitals of certain amounts as follows:

1. From each annual premium paid under the Act, the sum of 1s. 9d. is to be paid to the Victorian Treasurer into a fund—"The Motor Car (Hospital Payments) Fund". The fund will recompense those public hospitals which set apart special accommodation for the treatment of persons suffering injuries arising out of the use of motor cars, as follows:

(*a*) Yearly interest and sinking fund for capital expenditure for the special accommodation.

(*b*) Towards maintenance expenses of patients in so far as these expenses are not met by payments made to patients by authorized insurers pursuant to the conditions of the Act and amounts paid by patients under the Hospitals and Charities Act. And then

(*c*) To other public hospitals maintenance expenses for patients injured by motor cars, in so far as these are not met as in (*b*) above.

No payments made as above shall reduce any liability under the Hospitals and Charities Act to any person in respect of maintenance or relief provided by the hospitals to a patient or any liability under this Act to any hospital of any insurer.

The Act also provides for payments to be made to all hospitals—public, intermediate and private—in respect of in-patients injured by motor cars up to £50, and if out-patients up to £5, provided that the amount so paid must not exceed one-fifth of the total amount paid (exclusive of costs) by the authorized insurer in respect of the fatal or bodily injury to any patient. Such payments will, however, be made only when an insurer accepts liability or negligence is proven.

Payments made in accord with this provision shall be at the rate of "expenses reasonably incurred", that is (*a*) public hospitals—the average daily cost as shown in the previous year's annual report of the Charities Board, (*b*) intermediate hospitals—at the scale approved by the Charities Board, (*c*) private hospitals—the recognized charge.

Out-patient expenses — reasonable expenses actually incurred.

In addition there is provision for the payment by insurers to medical practitioners, nurses, chemists and ambulance services in cases where injured persons receive emergency treatment or are conveyed in ambulances, namely:

Section 23.

(1) Where—

(*a*) (i) any legally qualified medical practitioner or registered nurse renders, or any registered pharmaceutical chemist supplies surgical dressings medicines or drugs for, emergency treatment in respect of fatal or bodily injury to any person caused by or arising out of the use of a motor car; or (ii) the person so injured is 'immediately after such injury conveyed in any vehicle the property of any body of persons corporate or unincorporate carrying on ambulance services otherwise than for private profit: and

(*b*) any payment is made (whether or not with an admission of liability) by an authorized insurer under or in consequence of a contract of insurance under this Part in respect of the death of or bodily injury to such person; and

(*c*) notice in writing of a claim under this section is given by such medical practitioner, nurse, pharmaceutical chemist or body to the authorized insurer within one month after the occurrence out of which the death or bodily injury arose—

there shall be paid by the authorized insurer (as the case may be)—

to such medical practitioner nurse or pharmaceutical chemist an amount (not exceeding in the case of any such person the sum of One guinea together with any travelling expenses reasonably and necessarily incurred) in respect of the emergency treatment so rendered or surgical dressings medicines or drugs so supplied; and

to such body an amount based on the distance such vehicle is required to travel, at the rate of One shilling and eightpence per mile but not being less than Ten shillings and sixpence in the case of any such person, in respect of the conveyance of such person—

and the liability (if any)—

of the owner or driver of such motor car in respect of such death or bodily injury; and

of such authorized insurer to such owner or driver in respect of the contract of insurance—

shall be deemed to be reduced accordingly to the extent of the amount required to be paid by the authorized insurer under this section.

(2) In this section "emergency treatment" means such medical or surgical treatment or examination by a legally qualified medical practitioner or a registered nurse as is immediately required as the result of any injury as aforesaid.

Payments to Hospitals.

In connexion with this Act an article which appeared in *The Hospital Magazine* of January, 1941, is of importance to all medical practitioners. In this article the Inspector of Charities states that:

> Section 22 . . . raises many problems and complicates the relationship of medical staffs to public hospitals and will lead to demands for classification of patients in the "intermediate" and "private" groups. Payments under this section, however, rest on acceptance of proof of liability; therefore, unless the injured persons are known at the time of admittance to have sufficient means or some other person arranges to meet the cost of treatment, admittance to "public" wards, in accordance with the present practice, remains undisturbed; moreover, in those cases where liability may be accepted at the time of admittance (or immediately thereafter) the probable period of treatment will be of importance in determining "financial" classification.

The following extract from instructions issued by the Charities Board is of general interest:

Use of "Public", Intermediate or Private Beds.

As payments by authorized insurers in accordance with Section 22 of the Act rest on acceptance of proof of liability, it may not be practicable to determine at the time admittance is sought whether the patient has or will have sufficient means available to meet the cost of service in an intermediate or private ward. Therefore, unless the patient (or husband, wife, parents, guardian, employer or others as the case may be) produces satisfactory evidence of ability to meet from private means intermediate or private patient fees and arranges to do so, admittance shall be to a "Public" ward bed. Those beds may be declared "Motor Car Accident Beds", and in the event of a hospital committee, in accordance with Section 22 of the Act, sending notice of a claim to an authorized insurer, the bed may at that time be declared "intermediate" or "private" (as the case may be) to enable the honorary medical officer concerned to submit a claim for fees.

Where at the time of admittance the patient (or husband, wife, parents, guardian, employer or others as the case may be) seeks and can pay for accommodation in an intermediate or private ward, an admittance may be made if such beds are available.

In the event of such beds not being available, the patient should be referred elsewhere if a bed can be obtained. If a bed cannot be obtained elsewhere and the case needs immediate hospital care, the patient may be

admitted and the bed declared "intermediate" or "private", at the discretion of the hospital committee or its authorized executive officer.

Where liability for fees is not accepted or not required to be accepted by an authorized insurer, patients (or those responsible for them) admitted to a "public" ward should be required to pay in accordance with their means.

Employees injured while on duty as a result of motor car accidents and who lodge claims for compensation under the provisions of the *Workers' Compensation Acts*, would appear to be ineligible to benefit under the *Motor Car (Third-Party) Insurance Act*.

Services of Resident Medical Officers.

The services of Resident Medical Staff, except in emergency pending arrival of the medical practitioner concerned, are not to be available to patients (or Medical Officers responsible for them) admitted to intermediate or private beds.

Where in emergency a Resident Medical Officer is authorized to attend a private or intermediate patient a fee of 10s. per attendance will be charged by the Committee to the private doctor and disposed of as the Committee directs.

It is thus apparent that apart from some contribution towards the cost of "special separate accommodation" for persons injured by motor cars and possibly some payment for "immediate emergency treatment or examination" and for ambulance transport and provision for an injured person to claim after the death of an owner or driver and to claim on a nominal defendant if the car cannot be identified, there is little to be gained by any section of the community and no real attempt to recompense the medical profession for services rendered to members of the community injured by motor cars.

MEDICO-POLITICAL.

THE annual meeting of the Queensland Branch of the British Medical Association was held at the British Medical Association Building, Wickham Terrace, Brisbane, on December 13, 1940, DR. J. G. WAGNER, the President-Elect, in the chair.

War Emergency Organization: Commencement of Practice during War Period.

The following by-law was unanimously adopted:

No practitioner should commence practice without purchase in an area where practitioners are on active service for a period of two years, or the duration of the war, whichever is the less, except by special permission of the Council of the Branch.

This by-law is intended to deal with the commencement of practice both as a specialist and in general practice during the period mentioned.

With reference to the implementing of this by-law, the following resolutions have been passed by the Council:

1. No member may commence practice in Queensland without application to the Council.

2. Members intending to commence practice must make financial arrangements with the representatives of members recently practising in that district, who are absent with the Navy, Army or Air Force.

3. Where the parties cannot come to an agreement, the Council will assist in the negotiations.

4. Should this assitance fail to secure a satisfactory agreement the Council will exercise its discretion.

5. The Council shall appoint a subcommittee to deal with these negotiations.

Memoranda.

(a) The first aim of the Council in accordance with the recently passed by-law is to protect the interests of the members absent on war service.

(b) It is not the primary concern of the Council to protect under the by-law the practices of members remaining in the district.

(c) Any member who has purchased the practice of a member who is proceeding on war service, is entitled to protection for a period of six months.

(d) In the case of medical men starting in general and specialist practice, the Council will also be guided by the public need in the particular area or specialty.

Medical Practice.

THE NEW ZEALAND SOCIAL SECURITY ACT.

FOR the information of members of the British Medical Association in Australia we publish hereunder *in extenso* the regulations recently made by the Government of New Zealand in connexion with the *Social Security Act*, 1938 (as amended by Part II of the *Finance Act*, Number 4, 1940).

REGULATIONS.

1. These regulations may be cited as the Social Security (Medical Benefits) Regulations 1941.

PRELIMINARY.

2. These regulations shall come into force on the day following notification in the *Gazette* of the making thereof.

3. In these regulations, unless the context otherwise requires,—

"Appointed date" means the date appointed by the Minister, pursuant to clause 4 hereof, as the date on and after which medical benefits in accordance with the said Act and these regulations will be available:

"The said Act" means the Social Security Act, 1938:

"Department" means the Department of Health established under the Health Act, 1920:

"Health district" or "district" means a health district constituted under the Health Act, 1920:

"Medical Officer of Health" means a Medical Officer of Health under the Health Act, 1920:

"Medical practitioner" means a medical practitioner registered under the Medical Practitioners Act, 1914:

"Minister" means the Minister of Health:

"Patient", in relation to any medical practitioner, means a person for whom that medical practitioner has agreed to provide medical benefits in accordance with these regulations (whether or not such person is receiving or has at any time received any medical treatment from that medical practitioner):

"Patients List" or "List", in relation to any medical practitioner, means the list, compiled in accordance with clause 14 hereof, of persons for whom that medical practitioner has agreed, in accordance with these regulations, to provide medical benefits:

"Visiting area", in relation to any medical practitioner, means the area within which, in the course of his ordinary practice, he is prepared to visit patients who are unable to consult him at his surgery.

COMMENCEMENT OF MEDICAL BENEFITS.

4. (1) The Minister shall appoint a date on and after which medical benefits in accordance with the said Act and these regulations will be available.

(2) Notice of the appointed date shall be published in the *Gazette* and in such other manner, in each health district, as the Minister thinks fit.

(3) Nothing in these regulations shall be construed to affect the power to make special arrangements conferred on the Minister by section 82 of the said Act, as extended by section 15 of the Finance Act (No. 4), 1940.

SCOPE OF MEDICAL BENEFITS.

5. (1) In accordance with the provisions of subsection (1) of section 13 of the Finance Act (No. 4), 1940, but subject to the provisions of the next succeeding subclause, there is hereby excluded from the expression "medical benefits" as used in these regulations all medical services that are within any of the following classes, namely:

(a) Medical services afforded in relation to maternity benefits under the said Act:

(b) The administration by medical practitioners of anæsthetics in any case where the medical practitioner by whom an anæsthetic is administered acts in assistance of or in collaboration with any other medical practitioner or a registered dentist:

(c) Medical services that involve the application of special skill and experience of a degree or kind that general medical practitioners as a class cannot reasonably be expected to possess.

(2) In a case of emergency any medical practitioner who has undertaken to provide medical benefits for any person in accordance with these regulations shall be required to render to that person whatever medical services are in the circumstance in the best interests of his patient, notwithstanding that any such services may be excluded by the last preceding subclause from the expression "medical

benefits" as used in these regulations. Any medical services so rendered in a case of emergency shall be deemed to have been rendered as medical benefits.

AGREEMENTS BETWEEN MEDICAL PRACTITIONERS AND PATIENTS.

6. No person shall be entitled to receive any medical benefits in accordance with these regulations, for himself or for any member of his family under sixteen years of age, or for any other person in respect of whom he is entitled to claim medcial benefits, unless and until:—

(a) He has made application in a form (hereinafter referred to as the Application and Agreement Form) to be provided for the purpose by the Department for the issue to him of a Medical Benefits Card;

(b) He has duly furnished the particulars required to be furnished by him on the said form; and

(c) The form has been duly completed by the signatures of the applicant and of a medical practitioner who thereby undertakes (in accordance with a provision to be included in the form) to provide medical benefits in accordance with these regulations for the applicant or other person named in the form as the person entitled to receive medical benefits.

7. (1) Application and Agreement Forms shall be obtainable on application at the office of any Medical Officer of Health or at any post-office.

(2) Separate forms shall be completed for each individual by or in respect of whom an application is made under this clause.

(3) Forms in respect of children under sixteen years of age shall be signed by the father or mother of the child or by some other person who for the time being has the care or control of the child.

(4) Where by reason of age or infirmity or for any other sufficient reason any person who is entitled to claim medical benefits is unable to make the required application, the application may be made on his behalf by any responsible person, who shall add to his signature words indicating that, in making the application, he is acting on behalf of the person named in the form as the person entitled to claim medical benefits.

8. (1) The applicant, having first ascertained (by personal inquiry or otherwise) that the medical practitioner of his choice is willing to agree to provide medical benefits for the applicant or other person entitled to receive such benefits (as the case may be) shall complete his part of the Application and Agreement Form and shall present it to the medical practitioner for completion by him.

(2) If the medical practitioner is willing to undertake to provide medical benefits for the applicant or other person named in the form as the person entitled to receive such benefits, he shall complete the form by inserting therein any particulars required to be furnished by him and by signing the same, and shall then forward the form without delay to the Medical Officer of Health of the district in which the patient resides.

(3) If the medical practitioner is not willing to undertake to provide the required medical benefits, he shall return the form to the applicant, who may present it to any other medical practitioner who is willing to undertake to provide such benefits.

(4) The signing of an Application and Agreement Form by a medical practitioner shall constitute an agreement between the medical practitioner and the applicant whereby the medical practitioner undertakes, if and whenever required so to do, to provide medical benefits in accordance with these regulations for the applicant or, as the case may be, for the child or other person on whose behalf the application is made.

9. Nothing in these regulations shall apply with respect to any medical services rendered to any person by any medical practitioner otherwise than pursuant to an undertaking entered into in accordance with the last preceding clause.

COMMENCEMENT OF OBLIGATION TO PROVIDE MEDICAL BENEFITS UNDER AGREEMENT.

10. (1) Where an Application and Agreement Form has been completed on or before the appointed date fixed pursuant to clause 4 hereof, the obligation of the medical practitioner to provide medical benefits in accordance with his agreement shall commence on the appointed date.

(2) In every other case his obligation to provide medical benefits shall commence on the date on which the form is completed by him.

MEDICAL BENEFITS CARDS.

11. (1) On receipt of an Application and Agreement Form, duly completed in accordance with the foregoing provisions of these regulations, the Medical Officer of Health shall forward to the applicant a Medical Benefits Card in a form to be provided by the Department for the purpose:

Provided that in any case where the medical practitioner, in completing the Application and Agreement Form, has furnished particulars for the computation of the mileage fees payable in accordance with these regulations, the Medical Officer of Health, before or after the issue of a Medical Benefits Card, may require the applicant to furnish any particulars that may be required for the purposes of such computation.

(2) If the Medical Officer of Health is satisfied that any Medical Benefits Card has been lost or destroyed or has become dilapidated, he may issue a duplicate card and, if in the circumstances of the case he thinks fit so to do, may charge a fee of 1s. therefor.

12. (1) Before providing any medical services for any person by or in respect of whom such services are claimed as medical benefits a medical practitioner may require the production of the appropriate Medical Benefits Card. If any person claiming medical benefits from any medical practitioner refuses or fails to produce the Medical Benefits Card when required so to do, the medical practitioner, if he has any reasonable doubt as to the right of the person to claim such benefits, may decline to provide any such benefits unless and until the card is produced to him.

(2) Where any person, being required by a medical practitioner to produce a Medical Benefits Card as evidence of his right to claim medical benefits for himself or any other person, fails to produce the card, a fee (for which a receipt shall be given by the medical practitioner) may be charged for any services actually afforded by the medical practitioner.

(3) Where any such fee has been paid in respect of a person who was in fact entitled to claim medical benefits from the medical practitioner, the amount of the fee may be recovered from the medical practitioner by the Medical Officer of Health if application for a refund of the fee, accompanied by the receipt or other sufficient evidence that the fee has been paid, is made to the Medical Officer of Health within fourteen days after the date of payment. Any fee paid to a medical practitioner and recoverable in accordance with this subclause may be recovered from him as a debt due to the Crown or may be recovered by deduction from any moneys payable to him out of the Social Security Fund.

(4) On the recovery, in accordance with the foregoing provisions, of the amount of any fee paid to any medical practitioner, the amount recovered, less a penalty of not more than 2s. 6d., shall be refunded to the person by whom the fee was originally paid.

13. The Medical Benefits Card shall be deemed to be the property of the Medical Officer of Health of the district in which the patient resides, and shall be produced to him by the holder thereof if and whenever that officer so requires.

PATIENTS LISTS.

14. (1) From the particulars supplied in the several Application and Agreement Forms received by him in accordance with the foregoing provisions of these regulations, the Medical Officer of Health in each district shall in respect of each medical practitioner who has undertaken to provide medical benefits for persons resident in the district, compile a list (hereinafter referred to as the Patients List) in which shall be entered, *inter alia*, the name and address of every person for whom the medical practitioner has undertaken to provide medical benefits.

(2) A copy of the list or of such of the particulars recorded therein as the Medical Officer of Health thinks necessary shall be forwarded by him to the medical practitioner concerned.

(3) The Patients List shall be amended from time to time, as occasion requires, by the addition of new names and by the deletion of the names of persons who have died or in respect of whom the agreement to provide medical benefits has been in any way terminated.

(4) In order that Patients Lists may be kept up to date, it shall be the duty of every person to whom a Medical Benefits Card has been issued:

(a) To comply with any request made at any time by the Medical Officer of Health for confirmation or correction or amplification of any of the particulars furnished in the Application and Agreement Form, with reference to any person to or in respect of whom the Medical Benefits Card has been issued:

(*b*) To give notice, in accordance with the requirements of clause 23 of these regulations, of any change of address, for a period exceeding three months, of the person to or in respect of whom the Medical Benefits Card has been issued:

(*c*) If the name of the person to or in respect of whom the Medical Benefits Card has been issued is changed, by reason of marriage or otherwise, to give notice of the change to the Medical Officer of Health, within one month after the date on which the change of name was effected.

(5) If a request made by the Medical Officer of Health in accordance with paragraph (*a*) of the last preceding subclause is sent by registered letter and no reply is received within six weeks after the posting of the letter, the Medical Officer of Health may, after giving to the medical practitioner concerned not less than fourteen days' notice of his intention so to do, remove from the appropriate Patients List the name of the person to whom the request relates. On the removal of any name from a Patients List pursuant to this subclause the agreement between the patient or the person claiming benefits on behalf of the patient and the medical practitioner shall be deemed to be terminated.

OBLIGATIONS OF MEDICAL PRACTITIONERS WHO HAVE UNDERTAKEN TO PROVIDE MEDICAL BENEFITS.

15. (1) It shall be the duty of every medical practitioner who has undertaken to provide medical benefits in accordance with these regulations:

(*a*) To provide and maintain suitable surgery and waiting-room accommodation for his patients:

(*b*) To be in attendance for consultation and the treatment of patients at his surgery or at any other place approved for the purpose by the Medical Officer of Health, at regular times of which notice shall be given by the medical practitioner on his professional plate or otherwise as the Medical Officer of Health may approve or require:

(*c*) In the case of any patient whose condition of health is such that it is impossible or inexpedient for him to visit the medical practitioner at his surgery, to visit and treat the patient at his home or elsewhere within the visiting area of the medical practitioner:

Provided that a medical practitioner shall not be obliged to visit patients in any hospital unless, in accordance with the rules of the hospital, he is entitled, whether or not he is a member of the staff, to visit his patients in the hospital or in the ward or part of the hospital where they are for the time being accommodated:

(*d*) To prescribe such drugs and appliances as are requisite for the treatment of any patient, and in any case where any such drugs or appliances are required before they can be conveniently obtained elsewhere, to supply such drugs and appliances if he is able to do so:

(*e*) To issue, free of charge, such medical certificates or recommendations as may reasonably be required by any of his patients for any of the purposes of the said Act, or for the purpose of obtaining any medical or other treatment that is not within the scope of the obligations of the medical practitioner:

(*f*) To keep records of the clinical history of his patients and of his treatment of them and, if a complaint against the medical practitioner is made by or in respect of any patient, to produce the records or any of the records relating to that patient to the Director-General of Health if required so to do by notice in writing under the hand of the Director-General:

(*g*) To answer, in writing if need be, all reasonable inquiries made by the Medical Officer of Health with respect to any prescriptions, certificates, or recommendations given or issued by the medical practitioner to or in respect of any patient under these regulations:

(*h*) Generally to comply with the requirements of these regulations in their application to him.

(2) Where any medical practitioner supplies any drugs or appliances in accordance with paragraph (*d*) of the last preceding subclause he shall be entitled to demand and accept from or on account of the patient an amount not exceeding the reasonable cost of such drugs and appliances, unless, in connection with pharmaceutical or other benefits under the said Act, arrangements have been made for the supply of such drugs and appliances at the cost of the Social Security Fund.

SERVICES OF DEPUTIES, ASSISTANTS, AND PARTNERS.

16. (1) Save as provided in this clause in respect of partners and assistants, all treatment in respect of medical benefits shall be given personally by the medical practitioner who has undertaken to provide such benefits except where he is prevented from doing so by urgency of other professional duties, temporary absence from home, or other reasonable cause:

Provided that if the practitioner is unwilling to render any particular service or class of service (not being a service or class of service excluded from the scope of his obligations by virtue of clause 5 of these regulations) he may make arrangements with another medical practitioner for the provision of such service by that practitioner as his deputy and on his behalf.

(2) A medical practitioner shall make all necessary arrangements for securing the proper treatment of his patients where he is unable to give treatment personally, and shall inform the Medical Officer of Health of any standing arrangements for that purpose, and he shall not absent himself from his practice for more than one week without first informing the Medical Officer of Health of his proposed absence and of the person or persons responsible for conducting his practice during such absence.

(3) A medical practitioner shall not, except as a matter of temporary arrangement, employ an assistant to attend his patients without the previous consent of the Medical Officer of Health to the employment of an assistant:

Provided that if the Medical Officer of Health withholds such consent, the practitioner may appeal to the Minister, whose decision shall be final.

(4) Before consenting to the employment by any medical practitioner of more than one assistant, the Medical Officer of Health shall obtain the approval of the Minister.

(5) A medical practitioner shall not, without the prior consent of the Minister, employ as his deputy or assistant any medical practitioner who, by reason of a determination given in accordance with section 84 of the said Act, is not entitled as of right to enter into any contract of service under the said Act.

(6) Having due regard to the welfare of patients, a deputy, with the approval of the Medical Officer of Health, may attend for the purpose of giving advice and treatment to patients at times or places other than the times or places at which his principal has undertaken to attend for the purpose of giving advice and treatment to patients.

(7) A deputy or an assistant, acting for a medical practitioner who has undertaken to provide medical benefits in accordance with these regulations, in addition to signing with his own name any medical certificate or prescription or any other document required or authorized by these regulations to be issued by his principal, shall insert therein the words "Acting for [*Name of principal*]" or words to the like effect.

(8) A medical practitioner who engages any deputy or assistant shall be responsible for compliance by that deputy or assistant with the requirements of these regulations.

(9) In the case of two or more medical practitioners practising in partnership or as principal and assistant, treatment may at any time be given by a partner or assistant to any patient on the list of the practitioner instead of by the practitioner in person if reasonable steps are taken to secure continuity of treatment:

Provided that the patient will be entitled to require the personal services of the practitioner who has undertaken to provide him with medical benefits unless that practitioner is unwilling to render the particular service required or is prevented from attending the patient for any of the reasons referred to in subclause (1) of this clause.

TERMINATION BY MEDICAL PRACTITIONERS OF AGREEMENTS TO PROVIDE MEDICAL BENEFITS.

17. (1) Any medical practitioner may give notice in writing to the Medical Officer of Health that he desires to have the name of any specified person removed from his Patients List.

(2) On receipt of any such notice the Medical Officer of Health shall inform the patient, or as the case may require, the parent or guardian or other person having the care or control of the patient, and shall advise him that application should be made (in a form to be provided for the purpose on the Medical Benefits Card) for the acceptance of the patient by another medical practitioner.

(3) The name of any person to whom a notice under this clause relates shall be removed from the Patients List, in accordance with the notice as from the date of his acceptance as a patient by another medical practitioner or on the expiration of the month following the month during which the notice is received by the Medical Officer of Health whichever is the earlier:

Provided that if, when any such notice would take effect in accordance with the foregoing provisions, any person whose name is to be removed from the Patients List is under treatment by the medical practitioner it shall be the duty of the medical practitioner to notify that fact to the Medical Officer of Health, and in any such case the notice of removal shall not take effect unless and until the patient is accepted by another medical practitioner or until the expiration of the month following the month in which the Medical Officer of Health is advised by the medical practitioner that the patient is no longer under treatment or in immediate need of treatment.

18. (1) If any medical practitioner who has entered into agreements to provide medical benefits in accordance with these regulations desires to terminate all those agreements at the same time he may do so by giving to the Medical Officer of Health due notice in writing of his intention so to do.

(2) Except as provided in the next succeeding subclause, not less than three months' notice shall be given of intention to terminate agreements under this clause, unless the Medical Officer of Health, with the consent of the Minister, agrees in any case to accept less than three months' notice.

(3) Where any notice of intention to terminate agreements under this clause is expressed to be given on the ground that material alterations in the rights or obligations of medical practitioners have been or are proposed to be made to the disadvantage of practitioners, the following special provisions shall apply:—

(*a*) If no notice or if less than one month's notice has been given by the Department to the practitioner of intention to make such change the agreements may be terminated at any time within fourteen days after the taking effect of the change:

(*b*) If not less than one month's notice has been given by the Department to the practitioner of intention to make such change the agreements may be terminated by a notice the period of which shall be not less than half of the Department's period of notice. Every notice given by a practitioner under this paragraph shall take effect on a date to be specified therein in that behalf.

19. Notwithstanding anything in the foregoing provisions of these regulations, a medical practitioner shall not be entitled, except with the consent of the Minister, to terminate any agreement thereunder at any time while an investigation concerning him is in progress or is pending under section 84 of the said Act.

Termination of Agreements by Patients.

20. (1) Any patient who has entered into an agreement with any medical practitioner under these regulations, or any person who has entered into any such agreement on behalf of a patient, may terminate the agreement in accordance with the following provisions of this clause.

(2) Where a new agreement has not been entered into with any other practitioner the agreement shall be deemed to be terminated on the expiration of fourteen days after the receipt by the Medical Officer of Health of a notice in writing of intention to terminate the agreement, accompanied by the Medical Benefits Card of the patient.

(3) Where a new agreement is entered into with another medical practitioner, the original agreement shall be terminated and the new agreement shall commence to operate in accordance with the following provisions, namely:

(*a*) Where the patient is resident within the visiting areas of both medical pracitioners, and both practitioners consent to the transfer of the patient in the form provided for the purpose on the Medical Benefits Card, the transfer shall be completed on receipt by the Medical Officer of Health of the Medical Benefits Card with the transfer duly recorded thereon:

(*b*) Where the patient is resident within the visiting areas of both medical practitioners, as aforesaid, but the original practitioner does not content to the transfer of the patient, the transfer shall be deemed to be completed on the expiration of the month following the month in which the Medical Officer of Health receives the Medical Benefits Card with the Form of Agreement thereon duly completed by the applicant and by the new medical practitioner.

(*c*) In any other case the transfer shall be deemed to be complete on receipt by the Medical Officer of Health of the Medical Benefits Card duly signed by or on behalf of the patient and by the new medical practitioner.

Special Provisions affecting Medical Practitioners who resume their Practice after Service with any of His Majesty's Forces.

21. (1) This clause applies only with respect to medical practitioners who, having been in practice in any locality, resume practice in the same or substantially the same locality after service with any of His Majesty's Forces, in connection with the present war, whether in New Zealand or overseas.

(2) Where any patient, having entered into an agreement with a medical practitioner under these regulations, desires to be transferred to the Patients List of a medical practitioner to whom this clause applies, at any time within four months after that practitioner has resumed practice, the transfer of the patient shall, whether both practitioners consent thereto or not, be completed on receipt by the Medical Officer of Health of the Medical Benefits Card of the patient, duly signed by the new practitioner.

Termination of Agreements by Minister.

22. Any agreement under these regulations may be at any time terminated by the Minister by not less than three months' notice in writing given to the medical practitioner concerned, in any case where the Minister has made or proposes to make special arrangements pursuant to section 82 of the said Act, as extended by section 15 of the Finance Act (No. 4), 1940, and the notice is expressed to be given on the ground that such special arrangements have been made or are about to be made.

Change of Residence of Patient.

23. (1) If any patient whose name is included in the Patients List of any medical practitioner removes from his place of residence, as shown on his Medical Benefits Card (whether permanently or with intention of being absent from that place of residence for more than three months) it shall be the duty of the patient or of a person having the care or control of the patient, forthwith—

(*a*) To notify the Medical Officer of Health of the date of removal and the new place of residence; and

(*b*) To forward the Medical Benefits Card to the Medical Officer of Health, for the recording thereon of the new place of residence, or for such other purpose as the case may require.

(2) If any change of residence to which the last preceding subclause relates is to a place beyond the visiting area of the medical practitioner, the agreement with that medical practitioner shall be deemed to be terminated and the name of the patient shall be removed from the Patients List of the practitioner on notice of the removal being given to the practitioner by the Medical Officer of Health.

(3) If any person fails to give any notice that he is required to give under this clause, he shall be liable to reimburse to the Social Security Fund any moneys which, by reason of his default, have been paid to any medical practitioner.

Remuneration of Medical Practitioners under Agreements to provide Medical Benefits.

24. Every medical practitioner who has agreed to provide medical benefits in accordance with these regulations shall be entitled to claim and receive from the Social Security Fund for every person for the time being on his Patients List a capitation fee at the rate of 15s. per annum.

25. (1) For the purpose of computing the amounts from time to time payable to any medical practitioner by way of capitation the Minister shall fix monthly or more frequent accounting periods, and shall fix an accounting date for each accounting period.

(2) The Minister may at any time and from time to time vary the accounting periods and the accounting dates fixed by him under this clause:

Provided that, except for the purposes of adjustment on the making of any variation, no accounting period shall exceed one month, and the interval between any accounting date and the next accounting date shall not exceed one month.

(3) Notice of the accounting periods and the accounting dates fixed pursuant to this clause shall be given in the *Gazette* and in such other manner, if any, as the Minister thinks fit.

26. (1) For every person on his Patients List on the accounting date of an accounting period the medical practitioner shall be entitled to receive for that accounting period a proportionate part of the annual capitation fee.

(2) The Medical Officer of Health shall, as soon as practicable after each accounting date, send to every medical practitioner who has agreed to provide medical benefits for

persons resident in the district a statement of the number of patients on his Patients List on that date, and, unless within ten days after the receipt by him of such statement, the medical practitioner objects thereto, by notice in writing given to the Medical Officer of Health, the statement shall, subject to the proviso hereto, be deemed to be correct:

Provided that, if the statement is not in fact correct, any necessary adjustments in the amount of the capitation fees paid or payable for any accounting period may be made in any later accounting period.

(3) The capitation fees payable to any medical practitioner for any accounting period shall be paid not later than one month after the end of the accounting period for which they are payable.

(4) Where by reason of the death of a medical practitioner or for any other reason all his agreements are terminated before the end of an accounting period a proportionate part only of the capitation fees shall be payable for that period.

CONDITIONAL RIGHT TO CHARGE FEES FOR SERVICES THAT ARE BEYOND THE SCOPE OF MEDICAL OR OTHER BENEFITS.

27. (1) Any medical practitioner who, having undertaken to provide medical benefits for any patient in accordance with these regulations, renders to the patient any medical services that are not within the scope of his obligations to provide medical benefits, may, subject to the provisions of this clause, charge a fee for any such services.

(2) A medical practitioner who has undertaken to provide medical benefits as aforesaid shall not be entitled to demand or accept a fee for any specialist services (being services excluded from his obligations by virtue of paragraph (*c*) of clause 5 (1) hereof) unless:—

(*a*) He has held hospital or other appointments affording special opportunities for acquiring special skill and experience of the kind required for the performance of such specialist services, and has had actual recent experience in performing services of a similar kind; or

(*b*) He has had special academic or post-graduate study of a subject that comprises the specialist services rendered, and has had actual recent experience in performing services of a similar kind; or

(*c*) He is generally recognized by other practitioners who have direct personal knowledge of his work as having special proficiency and experience in a subject or subjects that comprise the specialist services rendered by him.

(3) Before any medical practitioner affords to a patient who is entitled to receive medical benefits any medical services that in his opinion are outside the scope of the obligations undertaken by him and for which he proposes to demand or accept a fee, he shall—

(*a*) Inform the patient or some responsible person on behalf of the patient that the patient is not entitled to such services as part of his medical benefits; and (*b*) Obtain the concurrence of the patient or other person as aforesaid to his rendering the services.

(4) In any case where any such services are given the medical practitioner shall, within seven days after his demand or acceptance of a fee, notify the fact to the Medical Officer of Health in a form to be provided by the Department for the purpose.

(5) The provisions of subclauses (3) and (4) of this clause shall apply, with the necessary modifications, in any case where, in the course of providing medical benefits for any patient, a medical practitioner engages the services of any other medical practitioner as consultant, anæsthetist, or assistant.

MILEAGE FEES.

28. (1) In addition to the capitation fees payable in accordance with the foregoing provisions of these regulations, medical practitioners who undertake to provide medical benefits shall be entitled to claim and receive from the Social Security Fund mileage fees computed in accordance with the Schedule hereto.

(2) Except as provided in the Schedule hereto, mileage fees shall be payable in respect of every person for whom a medical practitioner undertakes to provide medical benefits.

(3) Such mileage fees shall be computed by reference to travelling distances (as defined in the Schedule hereto). Travelling distances shall in every case be determined by the Medical Officer of Health. Particulars of travelling distances, for the information of the Medical Officer of Health, shall be supplied in the first instance (in respect of each patient separately) by the medical practitioner, in spaces to be provided for the purpose on the Application and Agreement Form, or, in the case of a patient who is transferred from one medical practitioner to another, on the Medical Benefits Card of the patient.

(4) Where by any change of residence or other altered conditions the travelling distance in respect of any patient is affected or is likely to be affected it shall be the duty both of the medical practitioner and of the patient, if aware of the change, to inform the Medical Officer of Health.

(5) On becoming aware of any altered conditions as aforesaid, the Medical Officer of Health shall make any necessary alterations in the travelling distance and shall give notice of the alteration to the medical practitioner concerned, and no alteration in the travelling distance shall take effect until such notice is given.

(6) Mileage fees under this clause shall be payable for each accounting period fixed by the Minister for the purposes of clause 25 hereof, and the mileage fees for any such period shall be payable when the capitation fees for that period are payable.

29. (1) If any medical practitioner who has undertaken to provide medical benefits in accordance with these regulations is obliged to travel more than three miles in order to visit any patient elsewhere than in a city or borough in which the practitioner resides or has his main surgery, he may charge mileage fees at a rate not exceeding 3*s.* for each mile or part of a mile of any distance travelled by him (such distance being counted one way only) in excess of the travelling distance by reference to which the mileage fees payable to him from the Social Security Fund have been computed.

(2) Any fees chargeable under this clause may be recovered by the medical practitioner from the patient or from any person liable for his maintenance.

MISCELLANEOUS.

30. (1) Any person who is entitled in accordance with the said Act and these regulations to claim for himself or on account of any other person any medical benefits from any medical practitioner who has agreed to provide the same may make a complaint in writing to the Medical Officer of Health—

(*a*) If the medical practitioner who has agreed to provide such medical benefits refuses or fails to provide any such benefits when required so to do; or

(*b*) If, in the provision of any medical benefits, the medical practitioner or any deputy or assistant has displayed any culpable lack of skill or any negligence or lack of care in the performance of his duties.

(2) Unless in the opinion of the Medical Officer of Health the complaint is trivial, he shall refer it for investigation and report to a Committee appointed by the Minister pursuant to section 83 of the said Act for the purpose of hearing complaints.

(3) Any complaint that is regarded by the Medical Officer of Health as trivial shall nevertheless be referred by him to the Minister if the complainant so requires, and if the Minister is of opinion that the complaint is well founded and is sufficiently serious he shall refer it to the Committee appointed to hear complaints.

31. (1) If by reason of the culpable failure of any medical practitioner to attend and treat any person for whom he has undertaken to provide medical benefits in accordance with these regulations any expenses are incurred by or in respect of the patient in obtaining or arranging for other treatment, the Medical Officer of Health may, subject to the provisions of the next succeeding subclause, recover from the medical practitioner, by deduction from any moneys then or thereafter becoming payable to him under these regulations, the amount of any such expenses that in his opinion were reasonably incurred.

(2) No expenses shall be recoverable from a practitioner in accordance with this clause except with the authority of the Minister and upon the recommendation of the appropriate Committee appointed under section 83 of the said Act.

32. With the prior consent of the medical practitioners concerned, the Minister may at any time and from time to time publish in such manner as he thinks fit a list or lists of the names of medical practitioners who have entered into agreements under these regulations.

SCHEDULE.

MILEAGE FEES PAYABLE FROM SOCIAL SECURITY FUND.

1. For the purposes of this Schedule—

"Borough" includes a city, and also includes any road district situated in the County of Eden; two or more contiguous boroughs shall together be deemed to be one borough:

The expression "travelling distance", in relation to any person, means the distance, counted one way only by the most direct route, between the usual place of residence of that person and the nearest main surgery or place of residence of any general medical practitioner:

The expression "general medical practitioner" shall not include.

(a) Any medical practitioner who is not actually engaged in the private practice of his profession; or

(b) Any medical practitioner who by reason of any determination given in accordance with section 84 of the said Act is not entitled as of right to enter into any contract of service under the said Act; or

(c) In relation to the computation of the travelling distances of any specified medical practitioner, any other medical practitioner who may be specified in that behalf by the Medical Officer of Health acting with the authority of the Minister.

2. No mileage fees shall be payable in accordance with this Schedule:—

(a) In respect of any patient whose usual place of residence is within a borough (as hereinbefore defined) in which any general medical practitioner resides or has his main surgery; or

(b) In respect of any patient if the travelling distance in respect of that patient is less than three miles; or

(c) In respect of the first three miles of any travelling distance; or

(d) Where the travelling distance exceeds twenty miles, in respect of so much of the distance as exceeds twenty miles:

Provided that the Minister may in any case authorize the payment of mileage fees for the whole or any part of such excess, at a rate not exceeding the rate prescribed by the next succeeding clause, if he is satisfied that the patient in respect of whom the fees are payable is resident within the ordinary visiting area of the medical practitioner.

3. Except as provided in the last preceding clause, every medical practitioner who agrees to provide medical benefits for any patient in accordance with these regulations shall, on receipt by the Medical Officer of Health of the completed agreement, become entitled in respect of that patient to receive from time to time, in accordance with these regulations, mileage fees computed at the rate of 2s. per annum for each mile or part of a mile of the travelling distance:

Provided that in any case where, in the opinion of the Medical Officer of Health, any part of the route of travel between the surgery or residence of the medical practitioner and the residence of the patient is such as would involve unusually time-consuming or unusually expensive means of travel, the Medical Officer of Health may increase the rate of the mileage fees in respect of the whole or part of the travelling distance.

C. A. JEFFERY,
Clerk of the Executive Council.

Post-Graduate Work.

WEEK-END COURSE IN SURGERY AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that a course of instruction in surgery will be held at the Prince Henry Hospital, Little Bay, during the week-end May 10 and 11, 1941. The programme is as follows:

Saturday, May 10.

9.30 a.m.—In Ward 22—Cases of interest: The Director of Post-Graduate Surgery, Mr. H. R. G. Poate.
11.15 a.m.—"Ethyl Chloride and Ether Anæsthesia: Some Practical Difficulties and their Management": Dr. H. J. Daly.
12 noon.—"Empyema of the Thorax": The Dean of the Faculty of Medicine, Professor H. R. Dew.
2 p.m.—"Fluid Balance in Relation to Surgery": The Director of Post-Graduate Pathology, Dr. F. B. Byrom.
3 p.m.—"Minor Surgical Affections of the Anal Canal": Mr. H. C. Rutherford Darling.
4.15 p.m.—"The Prostatic Position Today": Mr. R. J. Silverton.
5 p.m.—"Caecostomy": Mr. Eric Fisher.

Sunday, May 11.

9.30 a.m.—"Deafness": Mr. Huff Johnston.
10.15 a.m.—"Diseases of the Skull": Dr. H. R. Sear.
11.15 a.m.—"Some Common Surgical Conditions of the Abdomen in Children": Sir Robert Wade.
12 noon.—"Avoidable Mistakes": Mr. T. M. Furber.

The above lectures will be held in the lecture hall. A second week-end course in surgery will be held at the Prince Henry Hospital during the week-end August 2 and 3, 1941, a programme in regard to which will be published later.

The fee for either of these courses will be two guineas, except in the case of medical officers of the defence forces on full-time service, who are invited to attend the whole or any part of either of the courses free of charge. Applications for registration, accompanied by a remittance for the amount of the fee, must be made to the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 55, of March 20, 1941.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Promotion.—Surgeon Lieutenant Francis Harrison Genge is promoted to the rank of Surgeon Lieutenant-Commander, dated 4th March, 1941.

Confirmation in Rank.—Surgeon Lieutenant (D) (on probation) Allen Walton Hexter is confirmed in the rank of Surgeon Lieutenant (D), with seniority in rank of 1st May, 1939.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Volunteer Reserve.

Appointment.—William Henry Roberts is appointed Surgeon Lieutenant (on probation), dated 15th February, 1941.—(Ex. Min. No. 18.)

AUSTRALIAN MILITARY FORCES.

AUSTRALIAN ARMY MEDICAL CORPS.

Northern Command.

First Military District.

To be Major (temporarily)—Captain R. A. Maxwell, 13th November, 1940.

Reserve of Officers.—The appointment of Honorary Captain H. L. Carruthers is terminated. *To be Honorary Captains*—Gregory Perry Hildebrand and Lansell Leonard Grimmett, 4th February, 1941, and 14th February, 1941, respectively.

Eastern Command.

Second Military District.

Major N. M. Gibson, O.B.E., from the Reserve of Officers (A.A.M.C.), is appointed to command a General Hospital and is granted the rank of Lieutenant-Colonel (temporarily), 30th December, 1940. Captain M. R. Lee is transferred to the Reserve of Officers (A.A.M.C.), 5th February, 1941. *To be Captains (provisionally)*—Maxwell Steven Truscott, 3rd February, 1941; John Robert Osborne Roger, 10th February, 1941; Eric Hilton Miles and Edgar John Hardcastle, 14th February, 1941.

Reserve of Officers.—To be Honorary Major—Honorary Captain G. H. Hair, 29th January, 1941. *To be Honorary Captains*—Bouverie Primrose Anderson-Stuart, 28th January, 1941; and Geoffrey Hagarty, 6th February, 1941.

Southern Command.

Third Military District.

Honorary Captain B. J. Robinson is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 30th December, 1940. Lieutenant C. G. B. Colquhoun is appointed from the Reserve of Officers and to be Captain (provisionally), 6th February, 1941.

Reserve of Officers.—The date of appointment of Honorary Captain R. Wall which appeared in Executive Minute No. 235 of 1940, promulgated in *Commonwealth Gabette* No. 1 of 1941, is amended to read 26th October, 1940. *To be Major (temporarily)*—Honorary Captain John Rhys Williams, 7th December, 1940. *To be Honorary Captain*—Norman Moir Simpson, 6th February, 1941.

Western Command.

Fifth Military District.

Captain H. M. Burns is appointed from the Reserve of Officers (A.A.M.C.), 23rd January, 1941. The following officers are appointed from the Reserve of Officers (A.A.M.C.) on the dates shown, and to be Captains (provisionally): Honorary Captains H. R. Nash, 23rd January, 1941; W91 L. F. Healy, 1st February, 1941; W93 H. R. Pearson and W92 P. C. Hogan, 10th February, 1941.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Albert Edward Kahn, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 17th February, 1941.

The following Flight Lieutenants are transferred from the Reserve to the Active List with effect from 17th February, 1941: C. V. W. Brown, E. R. Edwards, H. R. Hawkins, B. C. Pirie, H. W. R. Sharp and J. L. Williams.—(Ex. Min. No. 38—Approved 19th March, 1941.)

Correspondence.

THE FREEDOM OF THE MEDICAL PROFESSION.

SIR: I have just read with interest Dr. Arthur Brown's article "The Freedom of the Medical Profession" in the journal of March 15, 1941. I have read several articles by Dr. Brown on the same subject and I feel very definitely that we owe him our gratitude for so clearly and uncompromisingly defining what he considers the faults in our present system and the remedies for these faults. It is to be regretted that his work has not evoked more discussion—his is almost a voice "crying in the wilderness".

In opposing the scheme for national insurance, which was nearly forced on us before the war, we succeeded in preventing an entirely inadequate and unfair system coming into being, but we lost in that there was created in the public mind a feeling that our attitude was merely a negative one and that we had no solution to offer.

Dr. Brown is urging what should be obvious to anyone who cares to think about it, that we start planning now for the reconstruction of the medical services after the war. No one can say the present system is in any way perfect. It is unfair in many ways both to the public and to the profession. If we gave a fraction of the time we willingly give in improving our efficiency as doctors to improving the system under which we work, there would be evolved a medical service controlled by medical men and rendering an ever-improving service to the community.

In writing this letter I am not advocating the system which Dr. Brown has outlined, because there are many points in it with which I do not agree, but I most heartily agree with his contention that the matter is urgent, and the time for us as a profession to find the solution to the problem is now.

He who thinks that the high taxation now operating is going to be reduced when we have won the war is, in my opinion, foolishly optimistic. The revenue from this taxation will then be used for creating new social services and a medical service is going to be one of these new services. Let us be ready—not with simply opposing some half-baked scheme produced by politicians, but a really sound scientifically conceived scheme formulated by ourselves.

Yours, etc.,

FRANK CH. DE CRESPIGNY.

Ararat,
Victoria,
March 17, 1941.

SOME MEDICAL ASPECTS OF CRIME.

SIR: I cannot summon words adequate to express my surprise at the immodesty of my friend who, in patting his colleague Dr. S. F. McDonald on the back, uncovers so shamelessly the darker and dedecorous elements in his subconscious mind. I refer, of course, to the letter by Dr. Paul G. Dane, published in your journal on March 8, in which he joins with Dr. McDonald in decrying an article by Dr. Dark, designed to enlighten us upon a pressing social problem.

To call the criminal an enemy of society is, with due respect to Sir James Barrett, simply to give a dog a bad name. The criminal is frequently the product of society. And it is high time we medical men recognized that criminal conduct is a symptom of a disorganized personality structure. This disorganization is the result of mental conflicts, feelings of insecurity and various mental maladjustments in childhood. The unfavourable psychological effects of a disrupted home, of an over-protective mother or a domineering (and possibly drunken) father are frequently embellished by economic insecurity, the adolescents' legacy from monopoly capitalism.

Punishment is not numbered among the implements of the psychotherapist. It is the traditional cry of those who seek vengeance, however unconsciously, upon the offender. And although any stick will suffice to beat a dog, it is possible that punishment is the least effective way of dealing with criminals. To offset his crass belief in the efficacy of punishment I would draw Dr. Dane's attention to the problem of recidivism. In Britain, not many years ago, the prison statistics show that there were 2,899 male and 1,023 female recidivists. The 2,899 men had between them 46,075 convictions; the women 39,215. The men had cost the State £2,764,500 and the women £1,660,560. In other words, each male prisoner had cost the people of Britain £954 and each female £1,380. None of these offenders had served less than five sentences and some had been convicted as many as two hundred times. Four and a half million pounds of public money was thus spent on treating 3,922 individuals by punishment which, to use Dr. Dane's words, "is the only method of sure evolution". Surely, Dr. Dane, psychoanalysis would have been less expensive—and more effective!

It is difficult to understand why Dr. Dane, the director of the Melbourne Institute for Psychoanalysis, should imagine that treatment of the criminal in the true sense should be in a "kid glove drawing-room manner" or why a rational psychotherapy applied to criminals should "make the way of the evil-doers easier". Presumably he is not averse to giving psychological treatment to the psychopath with an unconscious wish to murder his father; but when the harassed salesgirl steals a pair of stockings he removes his kid gloves and calls for punishment.

The man who cries for the punishment of an offender is one who feels the subconscious urge to punish evil impulses in himself; and in thus making a scapegoat of the criminal he punishes his own repressed wickedness and thereby increases his self-respect. Custom may sanction such conduct in a judge; but in a psychiatrist so incongruous a procedure borders on indecency.

Catullus noted that:

> We all hug a delusion somewhere hidden
> Unconsciously protruding from the sack . . .
> We see the way the other fools are ridden,
> But not the madman perched on our own back.
>
> (Translation: J. Lindsay.)

It is this madman who makes us say and do so many foolish things, keeps us "umbilical to earth", tied to tradition and unresponsive to ideals. He makes us behave like donkeys and spurs us on to destruction. He condemns what he cannot understand. He cries aloud for vengeance. He demands the punishment fit the crime. His presence on the backs of ordinary folk has come to be accepted. For the psychiatrist to be so saddled is inexcusable.

Yours, etc.,

REG. S. ELLERY.

33, Collins Street,
Melbourne, C.1,
March 18, 1941.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Brandon-Jones, Mary, M.R.C.S. (England), L.R.C.P. (London), 1938, Flat 4, 13, Milson Road, Cremorne Point.

O'Donovan, Daniel, M.R.C.S., L.R.C.P., 1919, 280, George Street, Sydney.

Maloney, John Bede, M.B., B.S., 1940 (Univ. Melbourne), Lewisham Hospital, Lewisham.

Northcroft, Earle Fead, M.B., Ch.B., 1934 (Univ. Edinburgh), Edgecliff Gardens, 415, Edgecliff Road, Woollahra.

Stephen, John Norman Robertson, M.B., Ch.B., 1934 (Univ. Glasgow), c.o. Mr. L. Hanrahan, 8, Castlereagh Street, Sydney.

Aram, Alexander John Thomas, M.B., B.S., 1940 (Univ. Melbourne), Base Hospital, Wagga Wagga.

Flaum, Ernst, M.D., 1928 (Univ. Vienna), 12, Edwin Terrace, Gilberton, South Australia, also recommended and approved for registration in terms of section 17 (2) of the *Medical Practitioners Act*, 1938.

The following additional qualification has been registered:

Macindoe, Norman Macleod, Burwood (M.B., Ch.M., 1925, Univ. Sydney), D.O.M.S.

QUEENSLAND.

THE undermenioned have been registered, pursuant to the provisions of *The Medical Act*, 1939, of Queensland, as duly qualified medical practitioners:

Barnett, Alfred John, M.B., B.S., 1939 (Univ. Melbourne), District Hospital, Mackay.

Black, Robert Hughes, M.B., B.S., 1939 (Univ. Sydney), District Hospital, Innisfail.

Tandy, Dorothy Rosemary, M.B., B.S., 1939 (Univ. Sydney), District Hospital, Innisfail.

Woolcock, William John Patrick, M.B., B.S., 1940 (Univ. Sydney), General Hospital, Toowoomba.

Obituary.

CLIVE WENTWORTH THOMPSON.

WE regret to announce the death of Dr. Clive Wentworth Thompson, which occurred on March 26, 1941, at Ashfield, New South Wales.

FRANK EDGAR WALL.

WE regret to announce the death of Dr. Frank Edgar Wall, which occurred at Sydney on April 1, 1941.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Leventhal, William Alexander, M.B., B.S., 1940 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Waddington, Austin Lawrence, M.B., B.S., 1933 (Univ. Sydney), Waterfall Sanatorium, Waterfall.

Arnott, David Bruce, M.B., B.S., 1940 (Univ. Sydney), 32, Campbell Parade, Bondi.

Medical Appointments.

Dr. I. W. MacNaught has been appointed Temporary Honorary Relieving Assistant Orthopædic Surgeon at Royal Alexandra Hospital for Children, Camperdown, New South Wales.

Books Received.

"Outlines of Industrial Medical Practice", by H. E. Collier, M.D., Ch.B.; 1940. London: Edward Arnold and Company, Limited. Demy 8vo, pp. 446. Price: 21s. net.

"Fractures and other Bone and Joint Injuries", by R. Watson-Jones, B.Sc., M.Ch.Orth., F.R.C.S.; Second Edition; 1941. Edinburgh: E. and S. Livingstone. Super royal 8vo, pp. 736, with 1,050 photographs, X rays and diagrams. Price: 50s net.

"Third Addendum to the British Pharmacopœia 1932", published under the direction of the General Council of Medical Education and Registration of the United Kingdom; 1941. London: Constable and Company Limited. Demy 8vo, pp. 40.

"Hospital Formulary and Compendium of Useful Information"; 1941. Berkeley and Los Angeles: University of California Press. Foolscap 8vo, pp. 275. Price: $2.00 net.

Diary for the Month.

APR. 8.—New South Wales Branch, B.M.A.: Organization and Science Committee.
APR. 8.—Tasmanian Branch, B.M.A.: Branch.
APR. 9.—Queensland Branch, B.M.A.: Brisbane Women's Hospital Clinical Society.
APR. 10.—Victorian Branch, B.M.A.: Ethics Committee.
APR. 15.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 16.—Western Australian Branch, B.M.A.: Branch.
APR. 17.—Queensland Branch, B.M.A.: Ipswich Hospital Clinical Society.
APR. 17.—New South Wales Branch, B.M.A.: Clinical.
APR. 17.—Victorian Branch, B.M.A.: Executive.
APR. 18.—Queensland Branch, B.M.A.: Council.
APR. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 23.—Victorian Branch, B.M.A.: Council.
APR. 24.—Queensland Branch, B.M.A.: Brisbane Hospital Clinical Society.
APR. 24.—New South Wales Branch, B.M.A.: Branch.
APR. 24.—South Australian Branch, B.M.A.: Branch.
APR. 25.—Tasmanian Branch, B.M.A.: Council.
MAY 1.—South Australian Branch, B.M.A.: Council.
MAY 2.—Queensland Branch, B.M.A.: Branch.
MAY 6.—Queensland Branch, B.M.A.: Post-Graduate Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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